

DISEASES AND PARASITES OF POULTRY

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cluster of many yellow vascular spheres which vary in size from that of the yolk in a normal egg down to a size which is not visible to the unaided eye. Each sphere is a more or less developed ovum or yolk enclosed in a thin membrane, and independently attached to the ovary by a very slender stalk. In the ovary of a non-laying hen the ova are all small, perhaps none being more than $\frac{1}{4}$ inch in diameter. The number of ova in a normal ovary is much greater than the number of eggs produced in the lifetime of a hen. Pearl (1912) made counts of the ova and ruptured follicles in 13 hens and reported numbers ranging from 914 to 3605. It does not appear that egg production is ever limited by the number of ova present.

The oviduct is a convoluted tube which, in a laying hen, fills a large part of the left half of the abdominal cavity. It is suspended from the dorsal body wall by a membranous ligament, and it lies above the abdominal air sac. The oviduct may be divided into five parts, primarily on the basis of function, though histological observations also lend support to the customary division. The five parts are (1) the funnel or infundibulum, (2) the albumen-secreting section or magnum, (3) the isthmus, in which the shell membranes are formed, (4) the shell-gland or uterus, in which the shell of the egg is formed, and (5) the vagina which opens into the cloaca.

In the functioning condition, the oviduct is often as much as 30 inches long and it is capable of considerable distention. It is well supplied with muscular tissue so that the egg is carried along during the process of its formation by peristaltic contractions. In the non-laying hen or in the pullet not yet ready to lay, the oviduct is very much smaller, perhaps not more than 4 or 5 inches in length.

The fully formed egg is normally retained in the uterus for several hours before being laid and while in this position it may be located by touch or palpation from the outside.

The Endocrine Glands—In the fowl as in other animal organisms, there are glands of internal secretion which have no excretory ducts but which secrete, into the blood or lymph substances which affect other organs of the body. These secretions are known as hormones, some of which are

PREFACE TO THE FIFTH EDITION

The preparation of this book was originally undertaken in an attempt to point the way toward a reduction in mortality among poultry flocks, which number more than 3,000,000, and which contribute directly to the income of about 70 per cent of the farm families in the United States. We have earnestly endeavored to present material which is scientifically accurate, and yet not so technical as to be difficult to understand and put into practice. Important advances in poultry disease research have made succeeding editions necessary.

Establishment of the Regional Poultry Research Laboratory, which opened in 1939 at East Lansing, Michigan, is evidence of the growing recognition of the importance of poultry disease research. A continuing program of such research is essential to the practical solution of new problems which are constantly arising to challenge the skill and the economic existence of flock owners.

It is our belief that the health and efficiency of any flock depend not only on the eradication and control of transmissible diseases, but even more on soundness of body and inherited vigor, on adequate nutrition in the sense of complete rations which are reasonably well balanced, and on suitable environment including physical and sanitary protection. It is our further belief that the successful control of most diseases and parasites of poultry depends on strict adherence to the principles of hygiene and sanitation and good management. New medicinal agents will continue to prove very helpful, but they should not be looked upon as substitutes for good management. We have tried to convey to the reader our conception of what these beliefs involve by emphasizing their relation to the problem of prevention and



FIG 10

FIG 11

FIG 10 —Pulling a few scutlers at the site of the incision

FIG 11 —Drawing the skin to one side preparatory to making the incision

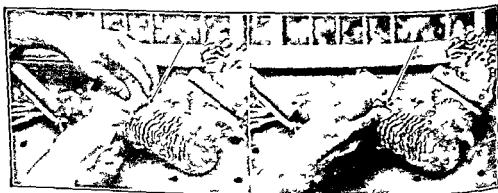


FIG 12

FIG 13

FIG 12 —The incision is made between the last two ribs

FIG 13 —A spreader is inserted and the incision enlarged



FIG 14

FIG 15

FIG 14 —Tearing the abdominal membranes to expose the testicle

FIG 15 —A close-up view of the incision and the testicle

control as it is discussed in connection with specific diseases and parasites

For the benefit of students of the subject we have selected from among more than 8000 literature references which were consulted something over 800 which in our opinion will be most helpful. Those which contain carefully selected or fairly complete bibliographies have been indicated by an asterisk (*)

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small probe which has a short tearing hook on one end and a spreader for holding the ribs apart after the incision has been made and a remover (several different styles are available) for taking out the testicles.

Good light is essential and if the weather is not too warm the operation is best performed in bright sunlight. Strict cleanliness should be observed even though the fowl is very resistant to the ordinary pus-forming organisms.

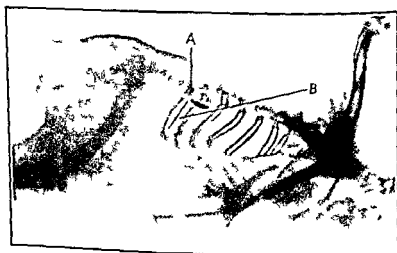


FIG 90.—Radiograph of a living cockerel. The testicle has been sketched in at A. The line of incision is indicated at B.

The incision is made between the last two ribs just below their connection with the backbone in an area that is covered by the thigh muscles when the bird is in a normal position. In order to expose the site of the incision and to keep the thigh muscles out of the way so that they will not be injured the bird must be stretched out with the legs extended and well up. Weighted cords looped about the legs and wings or straps fastened to a board may be used for this purpose. If the head is covered the bird will be less likely to struggle facilitated by using a sloping board to enable the operator to approach the

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prevent the bird from becoming a "slip." It is therefore important that the jaws of the remover make perfect contact. Care must also be taken not to rupture any of the primary blood-vessels or internal hemorrhage will result in death before the bird is removed from the operating table.

The remover is carefully worked over the testicle and so manipulated as to enclose the entire organ without including the artery. The testicle may then be drawn out with a slight twisting motion. If bleeding is at all profuse, the blood may be removed by inserting small pledgets of absorbent cotton held in forceps or tweezers, and removing them as soon as they become saturated.

After the testicles and all cotton or other foreign matter have been removed, the spreader is taken out and the skin allowed to slip back over the incision. The fowl may then be removed from the table, holding it carefully but securely so that it will not struggle, and placed in a clean coop or small yard where, with the other operated birds, it can be given food and water, and where it can be kept quiet for a few days. It is best to let the young capons rest on the floor or ground for a few nights, as the less flying and jumping they do until the wound is healed, the less likely is secondary hemorrhage to occur. The fowls seem to suffer very little inconvenience from the operation, and are usually ready to eat and drink as soon as released.

During the first few days they should be examined occasionally for "wind puffs," which sometimes develop from the accumulation of air under the skin. If large puffs are found they may be incised to release the air. The original incision should be entirely healed in from two to three weeks' time, and no further trouble is likely to result.

Synthetic estrogens closely related chemically to the female sex hormone are now widely used to bring about pseudo-caponization of cockerels. A 15-milligram pellet of stilbestrol implanted subcutaneously, will cause a cockerel to assume the appearance of a capon within ten or fifteen days. There is normally an immediate increase in feed consumption and a greatly increased deposition of fat.

Ovariotomy, or the unsexing of pullets, is not often prac-

Diseases and Parasites of Poultry

Chapter 1

THE MORTALITY PROBLEM

DISEASES and parasites continue to exact an annual toll from the poultry industry which has been estimated at more than \$250,000,000. This is a direct result of the unnatural but economically necessary increase in the size of poultry flocks, of the concentration of large numbers of fowls on relatively limited areas, and of the widespread interchange of baby chicks and breeding stock between different parts of the country.

Most of the economically important diseases of poultry are infectious and contagious. They are readily transmitted from fowl to fowl and from flock to flock, though sometimes by widely varying means.

In order to reduce or prevent mortality losses the poultryman needs to know:

1. How to avoid disease outbreaks by proper methods of management,
2. How to recognize disease outbreaks when they occur, and
3. How to obtain a correct diagnosis and specific directions for control.

Research has been slowly but definitely pointing the way to effective disease control and, because new diseases may be expected to appear from time to time, research must be continued in order to provide new answers which the industry will surely need.

Accomplishments of the Past.—There is much encouragement to be found in a comparison of present knowledge regarding disease control with the meager information available up to about 1910.

Pullorum disease then took its toll almost unchecked because the application of the agglutination test for the removal of carrier hens was not yet worked out. The whole-blood

treatment The feathers should be pulled from the area of the wound and the entire region should then be cleansed thoroughly with clean warm salt solution (2 level teaspoonfuls of salt in 1 quart of water) followed by a warm antiseptic solution, such as 1 per cent lysol

If the wound is extensive enough to require suturing, ordinary silk thread or small size chromic catgut may be used Needles and suture material should be placed in the antiseptic solution for a few minutes before being used After soaking in the solution for a short time, catgut becomes pliable and easily handled Individual, rather than continuous sutures are preferable, as they can be placed in just the right positions for most effectively closing the wound If deep sutures are required they should be made with catgut, as they will be gradually absorbed in from ten to twenty days and do not require removal Skin sutures may be of either silk or catgut Care should be taken not to draw the sutures too tightly Otherwise they may cut through the tissue and be of no value

After the wound has been closed, it should be sprinkled with a dusting powder such as iodoform, boric acid, or a mixture of sulfathiazole and sulfanilamide in equal parts

Bumblefoot.—An abscess of the foot is commonly called bumblefoot The primary cause is a bruise or cut on the bottom of the foot through which nonspecific infective organisms gain entrance A nutritional type of bumblefoot may result from a deficiency of vitamin A The condition is often not discovered until the fowl becomes lame Recovery may occur without any treatment, but it may be hastened by removing the scab which normally is found on the bottom of the foot, and pulling or pressing out the pus cores which are usually present If the infection has not become generalized, recovery may be prompt and complete In more severe cases it is necessary to cut or scrape out the accumulated material and, after thorough cleansing of the wound, to place the bird in a clean coop by itself where it can be kept quiet until it has recovered Bandaging of the foot will aid in keeping the parts clean One or two applications of antiseptic solution may be necessary at intervals of two or three days

stained antigen test which is now accepted as a routine procedure represents a remarkable advance in our ability to combat this disease

Coccidiosis made heavy inroads on many flocks because the life history of the causative organism and the numerous species with their varying habitats in the fowl were for the most part still to be discovered. The idea that young chickens could be exposed to a mild attack of coccidiosis in order to develop immunity against later unscheduled exposure would not have been accepted without the supporting scientific evidence from careful research

Untold thousands of birds were lost annually from going light before it was definitely established that this malady was frequently identical with tuberculosis and that the infected fowls could be identified by the tuberculin test. The simple procedure of disposing of all fowls at the end of their first laying year is now known to be sufficient to prevent all but very minor losses from this disease

Fowl pox took a yearly toll in mortality and decreased egg production. Owners of commercial flocks accepted with what grace they could the foregone conclusion that they would have chicken pox to contend with each year and merely hoped that it would not be complicated by the dreaded avian diphtheria which was not generally recognized as a manifestation of the same disease. What a contrast exists today when any flock owner can take advantage of modern vaccination methods with reasonable assurance that his losses from fowl pox will be virtually nil

Infectious coryza then known only as roup and colds was the despair of many a poultryman who watched its inroads year after year. With the knowledge that recovered birds are carriers it became a simple matter to isolate all young stock and to prevent any contact with older fowls. In this way the problem of controlling coryza became an easy one

Thousands of dollars and years of effort had been spent in attempting to solve the riddle of blackhead in turkeys but the essential points in its prevention and control which now seem so simple were still to be worked out

The nutritional disturbances to which fowls are so suscep-

the organism and young hatched from such eggs show evidence of the infection in a short time and many of them die. Some however survive the malady and grow to maturity with the infection localized in the ovary. In this manner, the disease is perpetuated from parent to offspring in a continuous cycle.

Marked differences have been noted in the virulence of individual strains of *S. pullorum*, some strains being highly virulent for both young and adults while others exhibited low pathogenicity for the young and were highly virulent for the adults and still others were relatively avirulent for both young and adult birds.

The resistance of *S. pullorum* to external influences such as cold sunlight and drying as well as to common disinfectants appears not to be marked. The organism apparently is somewhat refractory to putrefaction.

Occurrence—Pullorum disease is widespread throughout the world and is likely to be found wherever chickens and turkeys are grown. Because of its extreme importance in commercial poultry raising the disease has been extensively studied in order to develop effective methods for its eradication and control.

Symptoms—The symptoms displayed by affected chicks and poults are not constant. Some die so soon after hatching that no symptoms of any kind are shown, while others present a picture of general dejection. They stand "huddled up" as if cold, are droopy, and show little inclination to move. The eyes are closed and the affected birds frequently emit peculiar, squeaky chirps, as if in pain. In many there soon appears a whitish, pasty diarrhea and the vent is smeared with fecal material. There is little or no appetite, and irregular, labored breathing is often noted. Stafseth and Johnson (1927) reported large losses among infected chicks in which the symptoms were great thirst, watery, brownish diarrhea, and "swelling up."

The death rate usually reaches its peak within five to ten days after hatching, and while the symptoms described above are frequently observed they do not constitute adequate grounds upon which to diagnose pullorum disease.

tible, especially the vitamin deficiencies, were almost entirely unknown, and the losses which they occasioned helped to swell the total mortality from unexplained causes.

Most assuredly there is encouragement to be found in the record of these advances and accomplishments in the control of poultry diseases, and they simply represent the more outstanding examples without in any sense being a complete list. With such a record of progress it is perhaps no fantastic hope to predict that equally successful methods of combating the serious disease and parasite problems of the present day will be forthcoming.

The Problems of Today.—Irrespective of the accomplishments of the past and of one's optimism regarding the future, there is still the important question of what to do about the problems which confront present and prospective flock owners today. The general disease problem has come about partly through the unnatural concentration of large numbers of fowls in limited areas, and partly through the intensive application of improved methods of selection, feeding, housing and management to the one objective of increased egg yields. In the race for high egg production, the importance of inherent vigor and vitality has too often been overlooked.

From one point of view it might appear that the ability to lay 250 eggs in a year is circumstantial evidence of abundant vigor and vitality, irrespective of how that laying ability may have been brought about. Critical analysis, however, seems to indicate that it is entirely possible to throw the organism out of balance by stressing some one point in the selection program. A hen that has been bred to lay will continue to lay as long as it is physically possible for her to do so, even to the extent of drawing on her own body tissues to the physiological limit for the necessary protein, energy, vitamins and minerals to be used in egg formation. Such a hen is certainly "out of balance" in the struggle for existence, and must have man's artificial protection if she is to survive. The trouble may be that man has not learned as much about the needed protection (physical, nutritional and sanitary) as he has about the development of hens with the urge to lay. Of course, there are hundreds of thousands of flocks which are still far below the physiological limit, but wherever high-

bits of down, also dust particles carrying the organisms and are thus exposed to infection before leaving the incubator. Newly hatched birds are extremely susceptible to pullorum infection and the hatching of eggs from infected and non infected hens in the same incubator or even in the same room is obviously a dangerous practice.

The work of Hinshaw, Upp and Moore (1926) indicated that comparatively few microorganisms are present in the air of thoroughly cleaned and disinfected incubators. As hatching begins, however, the number increases rapidly and it was logically suspected that if *S. pullorum* is present it might thus be readily spread at hatching time among the chicks in the incubator. Studies proved this actually to be the case and means of suppressing such spread were sought. The possibility of fumigating incubators with germicidal agents received the attention of many workers and favorable results were obtained by using formaldehyde gas. Various methods of liberating the gas from formalin have been devised but the two methods most widely used are (1) the potassium permanganate and (2) the cheesecloth method.

Briefly the potassium permanganate method is employed by adding 17.5 grams of potassium permanganate to 30 cc of formalin for each 100 cubic feet of incubator space. The correct amount of permanganate crystals is placed in a large enameled pan which is then placed in the middle of the incubator. The calculated amount of formalin is then added and the door of the machine quickly closed. The door of the incubator should be kept closed for at least three hours to obtain maximum results. After the gas in the machine has been freed the chicks are removed and placed in clean boxes or quarters. Three fumigations twelve hours apart during the hatch are recommended with the first being made shortly after the first chicks have emerged from the egg.

The cheesecloth method is effected by dipping pieces of cheesecloth one yard square in formalin and suspending these wet pieces of cloth in the incubator near the fan. The amount of formalin recommended is 20 cc for each 100 cubic feet of incubator space and the cloths should be sufficiently wrung out as to be wet but not dripping. The treated cheesecloth is left in the machine for at least three hours at which time

producing stock is being distributed more widely and more rapidly than information on how to feed and manage it (and a willingness to apply that information), there is certain to be trouble of the sort suggested

The flock owner who is producing broilers for market is quite properly interested in securing rapid growth, for it is well known that the most efficient utilization of feed is made during the early growth period, but it does not necessarily follow that pullets which are reared under similar conditions will make the most profitable layers. Neither is it safe to assume that the groups or the individuals which make the most rapid growth are the most desirable specimens to save for breeding purposes except in the sense of probable transmission to their offspring of the characteristic of rapid growth. This condition which is so desirable from the viewpoint of broiler production, may perhaps be gained only at the expense of a shortened life span. Clear-cut evidence on this point is not yet available.

Health and physical efficiency in any flock are dependent upon (1) soundness of body and inherited vigor (2) adequate nutrition in the sense of complete rations which are reasonably well balanced, (3) suitable environment, including physical and sanitary protection, and (4) the eradication and control of transmissible diseases.

WHAT THE DEATH LOSS IS

Even a very casual inspection of the available reports of mortality in commercial and farm flocks and in egg laying contests will be sufficient to convince anyone that the loss of fowls by death often assumes serious proportions. Careful scrutiny of the records brings out the much more significant fact that the average death loss expressed as a percentage, has increased markedly during recent years. Furthermore, there is little evidence yet available to indicate that any marked improvement in the situation has begun to occur.

Mortality in Contest Flocks—The records of egg-laying contests probably constitute the most extensive source of accurate data on mortality in mature flocks, and it is enlightening, though not very encouraging, to review these records

parrot may be infected by human tubercle bacilli. Hogs are very susceptible to avian tuberculosis and heavy losses have resulted from the infection in swine because of condemnation of carcasses at the packing plants. Tuberculosis caused by the avian type of the organism has been reported in horses, sheep, cattle and swine. Rabbits are readily susceptible but rats and mice are relatively resistant to the infection. Man is apparently quite resistant and, although tuberculosis of avian origin has been reported in a few instances, the

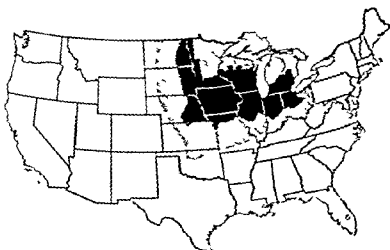


FIG. 31.—Showing the highest concentration of avian tuberculosis in 1926 (From the U. S. Department of Agriculture.)

percentage of infection in human beings is very low. Calves may become infected by association with tuberculous fowls, but they are far more resistant than swine, and the lesions are local in character.

Symptoms.—Fowls may become infected at any age, but because of the chronic nature of the disease, symptoms are not usually observed in birds less than one year of age. One of the first indications of tuberculosis in fowls is loss of weight ("going light"). As the disease progresses, emaciation becomes severe, until the affected bird weighs but a fraction of its normal poundage. In the later stages of the disease the combs, wattles, skin and mucous membranes of

One of the earliest reports of this sort is that of Dunning (1913)¹ who analyzed the mortality in ten years' egg-laying tests in New South Wales and found a death loss of 6.3 per cent a year for a total of 5448 hens. In three of the ten years the loss was less than 3 per cent, and in only one year did it exceed 10 per cent.

Card and Kirkpatrick (1919), in reporting the seventh Storrs competition, found a mortality of 15 per cent among 1000 pullets entered in that year, and called attention to the fact that this was a higher mortality than in any previous year of competition.

Anderson (1928) reported an average mortality of 25.4 per cent for the first five years of the New York State Egg Laying Contest at Farmingdale. In the sixth Farmingdale contest the mortality was still higher, being 28 per cent among White Leghorns and 45.6 per cent among Rhode Island Reds, these being the only breeds represented by 200 or more pullets.

Stafseth and Weisner (1931) reported the death loss among 10,000 pullets entered in eight successive laying contests in Michigan and showed that it averaged 19 per cent a year. Distribution of this mortality by months is shown in Table 1.

Since 1930 this contest has been started each year on October 1 instead of November 1. Corresponding data for the seven years, 1930-1937, as reported by Weisner (1937), are given in Table 2. The total number of pullets in the seven years was 7124. The death loss was higher than in the preceding eight years, and there was a rather marked seasonal shift in the mortality curve.

Alder (1934) gives the mortality percentages in each of seven successive contests in Utah as 13, 35, 33, 27, 18, 20 and 58, respectively. The excessively high figure in the last year is attributed in part to an outbreak of infectious laryngotracheitis shortly after the opening of the contest.

That this high mortality is not confined to the older plants is shown by the reports of the newly established New York State Egg Laying Contests at Stafford and Horseheads, in which the mortality during the first contest year, 1931-1932, was 24.7 and 26.1 per cent, respectively. Death losses at these

¹ A bibliography of literature cited is given at the end of each chapter.

These are grayish or yellow tumor-like swellings found in various organs and parts of the body, being most prevalent in the liver, spleen and intestines, less so in the bones, lungs, ovary, peritoneum and other structures. These tuberculous nodules vary in size from a pin-head to a small marble, and may be



FIG. 33.—A case of generalized tuberculosis showing nodules on the stomach, mesentery, small intestine and ceca. The liver and spleen from this case had the same general appearance as those shown in Fig. 32.

so numerous as to involve practically the entire organ, especially in the liver and spleen. When extensively diseased, the liver, as well as the spleen, is enlarged and the tubercles are readily observed upon examination. Nodules of various sizes may be found in the intestine, also in the peritoneum. When tubercles are present in the wall of the intestine, many of them open into the bowel and thus an active lesion may continuously discharge countless tubercle bacilli into the

TABLE 1—MONTHLY DISTRIBUTION OF MORTALITY IN THE MICHIGAN EGG LAYING CONTEST DURING THE YEARS 1922-1930 (DATA OF STAFFLETH AND WEISNER 1931)

Month	Total deaths each month	Per cent mortality each month *	Per cent of yearly total
November	62	0.62	3.3
December	137	1.37	7.2
January	136	1.36	7.2
February	127	1.27	6.7
March	221	2.21	11.6
April	235	2.35	12.4
May	216	2.16	11.4
June	166	1.66	8.8
July	208	2.08	11.0
August	124	1.24	6.5
September	143	1.43	7.5
October	100	1.00	5.3
Date unknown	23	0.23	1.2
Total	1898	18.98	100.1

* Calculated as a percentage of the total number of pullets at the beginning of the year

TABLE 2—MONTHLY DISTRIBUTION OF MORTALITY IN THE MICHIGAN EGG LAYING CONTEST DURING THE YEARS 1930-1937 (DATA OF WEISNER 1937)

Month	Total deaths each month	Per cent mortality each month *	Per cent of yearly total
October	115	1.61	6.1
November	108	1.52	5.7
December	127	1.78	6.7
January	130	1.82	6.9
February	140	1.97	7.4
March	184	2.58	9.7
April	174	2.44	9.2
May	192	2.70	10.2
June	197	2.77	10.4
July	204	2.86	10.8
August	158	2.22	8.4
September	161	2.26	8.5
Total	1890	26.53	100.0

* Calculated as a percentage of the total number of pullets at the beginning of the year

with a small hypodermic syringe a minute amount (1 small drop) into the skin of one wattle, the other being left as a control. The proper injection of tuberculin requires skill and care. The point of the needle must not be pushed through the skin because tuberculin deposited under the skin does not give the desired reaction. On the other hand, the injection must not be made too superficially as the skin is likely to rupture under the pressure.



FIG. 34.—The wattle test for tuberculosis. The swollen wattle is evidence of infection.

The results of the test are best read forty-eight hours following injection. Many reactions will appear within twenty-four hours, but some are slower in becoming evident, therefore in order that none be missed by reading too early, forty-eight hours should elapse after injection before final readings are made. A positive reaction consists of a doughy swelling of the injected wattle. They vary greatly in size and appear blanched as compared with the red color of the normal wattle.

The success of the test depends largely upon the skill of the operator and the potency of the tuberculin used. Occasionally an advanced case of the disease does not give a reaction, but such birds usually manifest clinical symptoms which are suggestive, and which should be considered in making the diagnosis.

A rapid agglutination test for the detection of tuberculosis in fowls has been reported by Moses, Feldman, and Maun (1943) and by Karlson, Zinober and Feldman (1950). Richey,

contests have since decreased to a low of 13.9 per cent in 1940-41, with an 11-year average of 18.1 per cent.

An occasional report of a more favorable nature is to be found as, for example, that of Dudley (1928) who calculated the death loss among White Leghorns, White Wyandottes and Rhode Island Reds in the Harper Adams Laying Trials (England) for the fifteen-year period 1912-1927, and found it to be 7.2, 6.8 and 5.7 per cent, respectively. An inspection of "The Register of Records in Laying Trials," issued annually since 1927 by the National Poultry Council shows, however, that there was a steady increase in losses by death among the 171,888 birds entered in recognized British laying trials through the year 1937. In 1926-1927 the mortality among all breeds in all trials was 6.6 per cent, but by 1936-1937 it had increased to 17.7 per cent.

According to the Council of American Official Egg Laying Tests, the death loss among 13,207 birds in all tests during 1947-48 amounted to 13.9 per cent.

Platt (1949) reported that mortality from all causes among 38,090 birds entered from 1937 through 1947 in standard egg laying tests in the United States averaged 17.0 per cent. There was a decrease from 21 per cent in the first two years to 14 per cent in each of the last three years of the 10-year period.

Brunson and Godfrey (1952) in reporting a 14-year summary of mortality in the Oklahoma egg laying test indicated a range from 26 per cent in the highest year to 14 per cent in the lowest, with an average slightly over 20 per cent.

Mortality in Commercial Flocks—It might be expected that poultrymen who raise their own pullets and who are in a position to give these pullets every opportunity by putting them in winter quarters at the right time and without ever leaving the farm would normally experience a low death loss in such flocks. The facts seem to be, however, that the loss of fowls by death is one of the important items of expense in producing eggs under commercial conditions.

App, Waller and Lewis (1918) reported a 7 per cent mortality among the flocks on 150 commercial egg farms in New Jersey in 1915-1916. Buster (1928) found a mortality of

tant means of dissemination but it emphasizes the need for properly disposing of dead birds.

Mortality — The mortality from avian tuberculosis varies greatly in different localities and flocks and depends upon several factors. Old infected hens kept in the flock from year to year failure to determine the cause of death of birds and the use of cure-alls favor the retention and spread of tuberculosis in the flock and thus raise the death rate. The insidious nature of the malady may allow it to gain such a foothold that before the disease is suspected the flock is badly infected.

Treatment — No medicinal agents have been found to be of any value whatever in the treatment of avian tuberculosis and the use of alleged cures only offers further opportunity for the disease to spread among the fowls.

Vaccination — In recent years the possibility of conferring protection by vaccination against tuberculosis in animals as well as man has received considerable attention. Various agents have been used but the one most extensively investigated is a culture of tubercle bacilli in which the virulence has been reduced by artificial cultivation on special media over a long period of time. This vaccine is called B. C. G. (*Bacillus* of Calmette and Guérin) after the French scientists who developed it. The results following its use are contradictory; some workers, particularly the French, have obtained a high degree of protection in their test subjects while other investigators obtained results varying from no protection at all to a moderate degree of immunity. Doyle (1930) vaccinated fowls with reduced doses of B. C. G. vaccine but upon subsequent artificial exposure of the birds no recognizable protection had resulted from its use and he concluded that the vaccine does not confer immunity against tuberculosis in fowls.

Prevention and Control — When tuberculosis is found in the flock measures for controlling the disease should be instituted immediately. If testing augmented by finding tuberculous lesions in birds examined *postmortem* has shown a large percentage of the flock to be infected the most practical procedure is to destroy and burn the visibly affected birds and dispose of the remainder. Diseased carcasses

24 per cent in 35 flocks having a total of 65,264 hens in the Petaluma district of California in 1926.

Scudder and co-workers (1931) found an average death loss amounting to 13 per cent among flocks totaling 271,337 hens in Oregon during the three-year period 1926-1928.

Thomas and Clawson (1933) reported a mortality of 20 per cent on 315,577 Leghorn hens kept in commercial flocks of about 1000 each in Utah during the three years 1929-1931. Misner (1932) found a mortality of 27 per cent on 108 New York State Poultry farms during the year 1930-1931. The average number of hens per farm in this group was 1454.

TABLE 3 —THE RELATION OF MORTALITY IN LAYING FLOCKS TO LABOR INCOME RECORDS OF 130 COMMERCIAL FARMS IN ALABAMA
(Data of Blackstone and Henderson, 1954)

<i>Mortality, per cent</i>	<i>No of flocks</i>	<i>Labor income per hour</i>
Under 10	28	\$2 03
10-19 9	58	1 34
20 29 9	27	88
30 or more	17	— 07
Av. 18% .	130	\$1 26

Jull (1934) cites a report of the death loss on 126 farms in San Bernardino County, California, from 1928 to 1933. The average number of hens per farm was 956 and the average mortality by years was 19, 24, 27, 37, 36 and 39 per cent.

Blackstone and Henderson (1954) reported that mortality amounted to 18 per cent on 130 commercial farms in Alabama, when calculated on the average number of layers during the year. Increasing mortality meant reduced labor income.

A very natural question in view of these facts is why and how poultrymen continue in business in the face of such astounding mortality costs. A partial explanation may be found in the tendency for some flock owners to develop an indifferent attitude toward the matter of death loss. To the beginner in poultry keeping, the loss of a few splendid pullets amounts to a tragedy, but to the man of more experience the matter of picking up and disposing of dead hens is likely to be looked upon as one of the chores. The magnitude

culm test, applied by a competent veterinarian offers the best means of early recognition of the disease.

FOWL CHOLERA

Cholera of fowls is an acute occasionally a subacute or chronic infectious septicemic disease characterized in typical cases by sudden death and a high mortality rate.

Cause—The microorganism causing fowl cholera is called *Pasteurella multocida*. It is one of the hemorrhagic septicemia group of organisms the members of which cause disease in several species of animals. *P. multocida* is not a very resistant organism and is quite rapidly destroyed by the disinfectants ordinarily used. When incorporated in soil or manure however it may live for as long as three months. Hendrickson and Hilbert (1932) found that *P. multocida* remained alive for eleven days in the carcass of a fowl kept at room temperature and for two months when kept at ice-box temperature. The organism is readily isolated from affected birds and when stained shows heavier absorption of the dye at the ends than in the middle thus causing it to be designated as 'bipolar'.

From widely separated outbreaks of fowl cholera Webster (1930) isolated organisms which produced one of three distinct types of colonies viz the 'fluorescent,' 'intermediate' and 'blue'. The fluorescent type was highly virulent and associated with severe outbreaks while the intermediate type possessed only a moderate degree of virulence. The blue colony type was still less pathogenic and was associated with less severe outbreaks.

Occurrence—Fowl cholera is widespread over the world and is very prevalent in some localities, being responsible for heavy losses. Records of outbreaks indicate that the disease is usually more prevalent during inclement weather and this may well be due to the closer contacts by fowls in such climatic conditions.

Susceptibility—The disease is highly infectious for chickens, geese, turkeys also for pigeons, ducks, pheasants and other wild birds. Hilbert and Witter (1936) reported outbreaks of fowl cholera in young ducklings five to ten weeks of

of the loss is realized only when careful records are kept and summarized, or when an epidemic of some sort results in the loss of a large number of fowls within a short time

Another reason is that on many farms a part of the mortality loss is avoided, and in a sense covered up, by the practice of culling. An expert poultryman soon learns to identify those fowls in the flock which are out of condition and which, if left to themselves, are likely to swell the next month's mortality figures. By disposing of such fowls while they still have some market value the owner of a large flock may avoid considerable financial loss at the time, but he is often shutting his eyes to the fundamental problem. He feels a false sense of security in his low mortality figures when the facts are that many of the fowls culled would not have lived through the year if left in the flock.

Mortality and culling not only cause a direct monetary loss amounting to the value of the fowls which die, but they result in a further indirect loss which may be even more costly in spite of being less apparent. Houses, equipment and labor sufficient to care for a flock of 4000 hens will be used with decreasing efficiency as the flock size is reduced during the year. This is partly obscured by the common practice of making calculations, such as average egg yield, on the basis of the average number of layers in the flock during the year, that is on the hen-day basis. A flock that has lost 50 per cent of the original number by death may thus show an average yield well above 200 eggs. If all such calculations were made on the basis of the number of hens and pullets at the beginning of the laying year, a more dependable measure of the real worth of the flock would be obtained. The best flocks are those which have a low death loss as well as a small number of fowls culled out as poor layers. They would be easily identified if all flock average egg records were reported as hen housed averages as is now done with laying contest records.

Mortality in Caged layer Enterprises — Increasing numbers of poultrymen, especially in the South and Southwest, are using cages for laying hens in preference to the more common method of floor management. Culling and replacement become a nearly continuous operation in such cases and may

The disease is commonly referred to as 'roup' by poultrymen because of the symptoms which are characterized by nasal discharge, swollen, tinged sinuses, swollen wattles and occasionally lower respiratory involvement. The exudate from affected birds has a typically offensive odor.

Postmortem Appearance. In fowls dead of the acute form of fowl cholera, the comb and wattles are dark in color, and there may be a sticky, tenacious mucus in the mouth and nasal passages. Reddish discoloration of the skin and breast muscles is a common but not constant finding. In general, the abdominal organs appear congested and darker in color than normal. Hemorrhages of various sizes are found on the heart, particularly around the coronary groove, and are also common in the abdominal membranes and in the subcutaneous and abdominal fat. The pericardial sac often contains an excess of yellowish fluid. The liver may be either very dark in color or lighter than usual, and its surface may be spotted with many small white foci. Severe inflammation and hemorrhages in the lining of the duodenum are lesions frequently present. The contents of this portion of the bowel are often tinged with blood, while in succeeding sections a yellowish sticky material is found. Pneumonia is occasionally encountered or the changes in the lung may be confined to congestion with numerous small hemorrhages. Cheesy, yellow deposits are often observed in various parts of the body, especially on the membranes of the air sacs, and about the intestines.

In chronic cholera, ovarian changes are not infrequently present and dried, cheesy, yellow yolk material is found free in the abdominal cavity or adherent to some organ. Beach (1922) concluded that rupture of the yolk might be caused by localization of the infection in the ovary. Shook and Bunyee (1939) observed an outbreak of fowl cholera in which proctitis was a very common lesion, ruptured yolks, salpingitis, hemorrhages of the ovary, and swollen wattles were also frequently noted.

Hoffman (1933) found that in 87 flocks suffering from the condition known as "ruptured yolk," 48 per cent of the birds examined were infected with *P. multocida*. If the birds died suddenly, no changes were present in the ovary, but if the disease

run to 7 or 8 per cent a month. Laurent (1955) reported an average death loss of 10 per cent of the average number of hens for the year on 22 farms for which complete records were available. Greene, Brooks and Niles (1937) reported a corresponding figure of 17 per cent for 33 cage layer operations in western Florida.

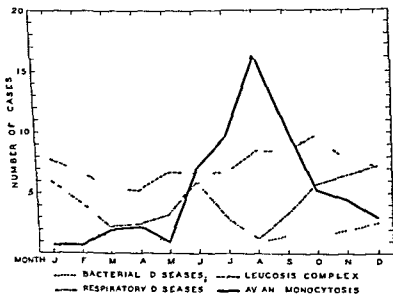


FIG 1—Seasonal variation in the incidence of poultry diseases (After Jungherr)

Mortality in Farm Flocks—Not so many data are available in the way of actual mortality records on farm flocks, but there seems to be no question that in many such flocks the death loss is a serious problem. One of the most favorable reports is based on the data of Winton and Canfield (1930), showing that the mortality in 331 flocks of the heavy breeds in Missouri averaged 9.7 per cent during the period 1919–1925 with a corresponding figure of 11.1 per cent for 207 flocks of light breeds. These 538 flocks contained 105,317 hens enough to make a representative sample.

Records of 224 farm flocks in Illinois in 1925 showed an

exposure to cold, or improper or sudden changes in the ration, the germs may be able to attack the body and induce disease. Hughes and Pritchett (1930) found that virulent cholera organisms when fed in capsules did not induce the disease but when instilled in the nasal membranes caused typical fowl cholera. From their studies of outbreaks they concluded that the more resistant birds were not affected, those less resistant were infected but recovered and became carriers while the most susceptible fowls died of acute, typical fowl cholera.

Pritchett, Benedictt and Hughes (1930), in studies of endemic cholera showed the reservoir or focus of infection to be apparently healthy pullets which had survived a previous outbreak. Pritchett and Hughes (1932), in later studies detected considerable variation in the virulence of strains of *P. multocida* isolated from different outbreaks. Some strains when introduced intranasally produced fatal cholera in about 40 per cent of the exposed fowls, but the organisms did not survive in the nasal cleft of resistant birds and did not spread to susceptible contact fowls. Other strains when similarly introduced failed to kill the exposed chickens but did survive in the nasal clefts and spread readily to susceptible fowls. From these studies it appears that the respiratory tract is the common portal of entry for *P. multocida* in fowls.

It has been shown that insects may also be a factor in the transmission of fowl cholera. Patton (1926) isolated virulent *P. multocida* from mites taken from a sick bird, and Skidmore (1932) demonstrated the ability of the common house fly to transmit the infection.

Introduction of the disease into the flock may be brought about by infected wild flying birds, particularly sparrows and pigeons, by the introduction of new stock, and probably on the clothing of human beings.

Mortality—The death loss from fowl cholera varies in different outbreaks and may range from only a few deaths to a mortality of 50 per cent or more.

Treatment—Many drugs have been tried as therapeutic agents but the results, in general, have been unsatisfactory. Delaplane (1945) suggested that sulfaquinoxaline in doses

average mortality of 13.2 per cent with a range of 5 to 50 per cent for individual flocks

According to Jull (1934), the mortality in demonstration flocks in various states, presumably for the year 1932, was 17 per cent in New Hampshire, 23 per cent in Massachusetts, 29 and 21 per cent for pullet and hen flocks respectively, in Delaware, and 37 and 38 per cent for pullet and hen flocks in Maryland

Chick Mortality —Although mortality among young chicks can be and often is just as serious a problem as it is with laying stock, the general situation with respect to chick losses is much more encouraging because of the clear cut and definite success of control measures. Mounting death losses among chicks were the rule a few years ago, but because they were concentrated in a period of a few weeks they were readily apparent and their seriousness was quickly recognized.

These losses led to "Grow Healthy Chick" campaigns and the adoption of rather rigid sanitation programs which have shown clearly that death losses among chicks can be greatly reduced. Judging from the results of sanitation plans which have been carried out in a number of states, it is entirely possible to reduce the total mortality, from hatching time to maturity, to something less than 10 per cent. It is probable that it will become increasingly necessary for a commercial poultryman who expects to continue in business to adopt a management plan which will insure a gross rearing mortality of not to exceed 10 per cent for the season.

Voorhies and Reid (1931) using data on the first fourteen days of the brooding period for over 6,000,000 chicks in the years 1927, 1928 and 1929, made an extensive study of daily mortality. Their data show a rapid rise to a sharply defined peak on the fifth day, with a rapid falling off from the sixth to the fourteenth day, at which time the rate of loss is down to the same level as on the first day. They concluded that there are indications of a typical curve depicting the chances of life for a baby chick during the first fourteen days of brooding, and that baby chicks have approximately 920 chances out of 1000 of reaching the fifteenth day of the brooding period.

Barrett (1929) studied the causes and distribution of mor-

under ordinary conditions. Transmission experiments carried out by these workers were totally unsuccessful. The disease was not contracted by healthy fowls through natural exposure during a period of several months, nor could it be produced by rubbing diseased tissues into the normal cloaca, the scarified cloaca, or the scarified cloaca treated with acetic acid. Feeding of the diseased tissue to fowls failed to produce the disease.

The disease is sometimes very resistant to treatment, but Goldberg and Benson, and Scherago reported that several cases recovered promptly after removing the necrotic tissue and washing the wounds with mild antiseptic solutions. Durant (1935) found that a majority of cases could be cured by three or four applications of a 3 per cent solution of chromic acid. Treatment consisted in gentle sponging of the affected parts with a cotton applicator at intervals of three or four days without removal of any of the diphtheritic membranes.

CANNIBALISM

Closely associated with egg bound and prolapse, because they are so often the inciting cause is cannibalism. While many flocks are never troubled in this way, others may be largely destroyed if the vice is not brought under control. The loss from this cause has become so serious in commercial flocks that many mechanical devices have been developed as aids in reducing the mortality. These range from vent shields, which protect the fowls on which they are placed, to guards fastened in the beak in such a way that the fowl cannot pick another. Some of these devices have been reported to be very effective when applied to laying fowls, but they are not so well adapted to young chickens. Temporary control can be effectively accomplished by cutting back the tip of the upper mandible of the beak to such a point that picking at another fowl is actually painful to the attacking bird. The beak grows out again, of course, in a short time. An alternative procedure is to sear the tip of the upper mandible with a hot soldering iron.

When cannibalism develops, as it often does, in chickens of brooding age, some other means of control must be adopted

tality among a total of 4806 chicks in seven hatches during the first eight weeks, and found a total loss of 11 per cent. He concluded that when both pullorum disease and coccidiosis are factors in the death loss, the mortality curve, plotted on a time base, is characteristically bi-modal. In the absence of coccidiosis it is mono-modal. He also found a relatively high initial mortality, unassociated with disease, which is in accord with the data of Voorhies and Read.

THE PROBLEM OF DISEASE CONTROL

If, as the records suggest, the increase in poultry mortality is closely associated with the increased use of intensive methods of rearing and production, and with the concentration of the poultry population in particular areas, a solution of the problem becomes of vital importance to every flock owner. It cannot be left to chance.

The problem is complicated by the fact that as a natural result of improved methods of feeding and management, and of selection and breeding for egg production, hens are expected to lay during a large part of the year instead of only during the spring and summer months. This means that many flocks have too little reserve vitality with which to fight an infection when it appears. If, as seems probable, still more attention is to be given to methods of breeding and management for increased egg production, the danger of loss from diseases and parasites is certain to increase at the same time unless very definite attention is given to means of controlling such troubles.

No individual can solve this problem alone, but each can do his part in hastening progress toward a practical solution. There is very great need for close coöperation among all groups which are in any way connected with the poultry industry. Flock owners, hatcherymen and feeding station operators have a common interest in healthy, vigorous stock. Scientific workers with an interest in poultry in the fields of bacteriology, immunology, parasitology, pathology, embryology, genetics, nutrition and poultry management must continue to unite their efforts to develop more effective methods of producing and maintaining such stock.

An excellent example of what a careful sanitation and quarantine program can accomplish is furnished by the experience of the Regional Poultry Research Laboratory at East Lansing, Michigan. Through a period of eleven years after the laboratory was established the stock was kept free of all diseases other than the one with which they were working, namely, the avian leukosis complex. The only exception was the infrequent occurrence of coccidiosis, and this disease was kept under control. During the eleven years they had no pullorum disease, no bronchitis, no colds or coryza, no cholera, no typhoid, no laryngotracheitis, no Newcastle disease, no fowl pox, no lice, no mites, no roundworms, no tapeworms. In other words, the sanitation procedures actually worked. Not until the spring of 1950 did any contagious virus disease, other than lymphomatosis, occur. In May of that year there was an outbreak of infectious bronchitis. The source of the infection was not positively determined, but circumstantial evidence pointed strongly to an unfortunate break in the sanitation procedures.

The essential points in the sanitation and quarantine plan in effect at the laboratory are

1. A "closed" breeding flock is maintained. Hatching eggs were purchased at the beginning, and no stock has been added since. This is highly important.
2. Quarantine measures are rigidly carried out, as indicated in some detail in the following statements.
3. No chickens from outside sources are accepted for diagnosis.
4. No one can go in the plant without passing through a locker room in which shoes and outer clothing are changed. Lunch buckets, watches, pocket knives and other personal items are passed through an ultraviolet sterilizing cabinet before being taken into the plant. Working clothes are furnished by the laboratory as a protection to the experiments being conducted.
5. The plant is divided as between isolation breeding flocks and those used for transmission experiments. Individual caretakers work exclusively on one side or the other. Furthermore, a change of shoes is made on leaving or entering each house.

- 6 No visitors are allowed on the plant at any time
- 7 Employees are prohibited from keeping chickens at home, simply as an added safeguard against introducing infection
- 8 All chicks are started in batteries and transferred to brooder house pens with wire panel floors
- 9 All buildings are screened against wild birds, flies and mosquitoes. Insects have been controlled also by the use of DDT, Lindane and pyrethrins in oil-base sprays
- 10 All feed is delivered in bulk, and elevated to bins in the feed house, so that neither truck nor driver is ever on the plant. Furthermore, the truck is washed before leaving the mill, and the driver makes no intermediate deliveries before reaching the laboratory. A similar type of small bulk delivery truck is used on the plant itself
- 11 The plant has its own water supply from a deep well and an enclosed water tower
- 12 All chickens are raised and maintained in complete confinement within screened buildings
- 13 The pens and equipment are cleaned regularly and thoroughly, and steam sterilized after each cleaning
- 14 The litter used in the laying and brooding pens was chosen because it is subjected to high temperatures during the manufacturing process
- 15 A vigorous campaign is maintained to keep out rats, mice, and other wild rodents
- 16 All refuse, litter, feces, and other such matter go to a compost pile in a remote corner of the plant. The composted material is removed by way of a back entrance about once in two years. Carcasses and offal are buried in a trench with quicklime. Those involved in autopsy are incinerated

Such a complete program would of course be difficult to carry out on a commercial basis, and it probably has no place in the management of farm flocks. But the principles on which it is based are sound, and many of them can be applied much more widely than at present with distinctly profitable results. Simpler programs have repeatedly been shown to be practical and highly effective. Most authorities

and experienced operators would agree on the following procedures, with certain variations and additions under special conditions.

1. Avoid the introduction of partly grown or adult stock. Breeders should maintain closed flocks. Others should make necessary introductions by means of eggs or chicks from best known sources.
2. Isolate and rear chicks away from adult stock. Grow chicks indoors, or provide enough land so that range-grown chicks do not occupy a given area more often than once every three years. If chicks from two or more sources are to be grown at the same time, isolate them from each other during the first two or three months.
3. Avoid traveling directly from the adult flocks to the chick range, if at all possible. If the business is large enough to make it practical, have separate caretakers for chicks and hens.
4. Keep visitors out of the houses and yards, especially those whose business takes them from farm to farm.
5. Maintain clean, sanitary quarters. Have a clean water supply. Use feeders which reduce waste to a minimum, and prevent contamination of feed by the chickens. Avoid spillage of feed where it will attract wild birds and rodents.
6. House pullets and hens separately. This not only is good management, but it protects the pullet flock from infections which may be spread by "carrier" hens.
7. If such diseases as fowl pox and laryngotracheitis are a problem on the farm or in the community, vaccinate all fowls on the premises, and continue to vaccinate each new crop of chickens year after year.
8. Follow procedures which will keep the fly nuisance at a minimum. In certain areas it may be equally important to control mosquitoes and other biting insects.
9. Keep the poultry areas sufficiently well fenced to prevent the escape of chickens to neighboring farms, and to prevent chickens from other flocks from gaining access to the premises.

10. If a breeding program is being followed on the farm, practice rigid selection in order to make use of high-viability families year after year.

Even under the best of conditions, and in spite of rigid sanitary procedures, disease will sometimes occur. When it does, the first step should be to get an accurate diagnosis if possible. With that accomplished, one can follow the best known control measures in combating the disease. Without it, one can only proceed blindly.

It is unfortunately true that there are still many cases which cannot be diagnosed with accuracy. Under such conditions the veterinarian often cannot help, the laboratory findings are negative, and the only certain fact is that the fowls are dead. This non-specific mortality is one of the problems still awaiting a solution.

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Chapter 2

THE NATURE OF DISEASE

DISEASE may be defined as any deviation from health whether it be a slight ailment, or one endangering life. Little understood for centuries, many disease processes have in recent times yielded to study and experimentation, from which the necessary knowledge has been gained to enable man to cope successfully with them. The modern approach to the solution of a disease problem necessitates the utilization of many sciences. Those ailments affecting poultry are no exception and the sciences of bacteriology, immunology, parasitology, protozoology and pathology have all contributed greatly to a better understanding of the many diseases and parasitoses to which fowls are susceptible.

DEFINITIONS OF TERMS

To promote a better understanding of some of the terms which must of necessity be used in any discussion of disease, and to explain briefly a few of the principles relating to pathological processes, there are given here some definitions and explanations which it is hoped will prove helpful. It is obviously impossible in a book of this scope to deal with more than a few of the important and fundamental principles.

Infection is the successful invasion of the body by disease-inducing agents such as bacteria, molds and viruses. The disturbance induced by such an invasion may be so temporary and slight as to produce only mild, transitory symptoms or it may be severe and prolonged, resulting in extensive alteration and discomfort, or even death.

Virulence refers to the degree of ability of a disease-inducing agent to set up a disturbance in the normal health of the invaded individual—it may range from slight to extreme.

Pathogenic also refers to the ability of an organism or other agent to cause disease but does not, in common usage, indicate the degree of invasive power.

Resistance may be defined as the opposition offered by the body of the invaded individual to any infectious agent. It may vary from only a slight resistance to complete protection.

Immunity denotes the condition of being refractory to a disease-inducing agent, and thus not susceptible to its pathogenic powers. Immunity is not an absolute state, but is subject to alteration by numerous influences such as dosage and virulence of the infectious agent as well as by the age and general well-being of the individual.

(a) Active immunity is that produced by the reaction of the body cells in response to the stimulus of an infectious agent—it is produced by the host itself. The development of immunity which follows vaccination of a fowl with fowl pox virus is illustrative—it has been engendered by the fowl itself.

(b) Passive immunity is that state of resistance conferred by the injection of serum from an individual which is immune to a particular disease, the body of the recipient having played no part in the production of the immune substances. Such immunity is usually of short duration.

(c) Acquired immunity is that acquired by an individual by any means. It may be active or passive and induced either by a previous attack of the disease or by the injection of causative agents or their products (vaccines, serums, etc.).

Etiology refers to the study of the causative agent of any disease process. This may include the determination of the nature of the offending organism or the factors favoring invasion or both.

Inflammation is the reaction of the body tissues to irritation and is characterized by heat, pain, redness, swelling and altered function. The suffix "itis" is often used to indicate inflammation of a specific part, *e.g.*, enteritis denotes inflammation of the intestines; cloacitis indicates inflammation of the cloaca.

Lesion is a term used to denote a visible morbid change in the structure, color, or size of an organ or part of the body. Some lesions are so minute as to require magnification for detection, others are readily visible and often extensive.

A *vaccine* is usually defined as a suspension of a living, disease-producing agent such as bacteria, or a virus. In

some instances, the virulence of the agent is reduced or attenuated by some means such as dilution, heat, biological methods, or chemicals to render its use in the field safe. Complete inactivation of viruses is sometimes necessary when they are used as immunizing agents.

A *bacterin* is a suspension of dead organisms, regardless of the means used to kill them. Bacterins have not, in general, merited extensive use in poultry practice.

A *toxin* is a poisonous substance produced by certain of the bacteria. Some toxins are only slightly virulent, others are deadly, even in minute amounts.

Antiserum is the blood serum of an individual which is immune to any specific disease-inducing agent.

Antitoxin is a specific type of antiserum and refers to the serum of an individual which is immune to a specific toxin. The body of such an individual has reacted to the presence of a toxin and produced protective substances or "antibodies" which are concentrated in the blood serum. When injected into a susceptible individual, antitoxin has the property of protecting against the toxin which was responsible for its production in the body of another. Antitoxins are specific and a given antitoxin will protect only against its specific toxin.

Agglutinins are but one of the several antibodies or protective substances produced in the body of an individual in response to the irritation or stimulus of infection. With but few exceptions they are specific, and this forms the basis of their detection by means of the agglutination test. This test and its practical application will be discussed in a later chapter.

THE NATURE OF DISEASE-INDUCING AGENTS

Bacteria.—Bacteria are one-celled, microscopic organisms occurring everywhere in Nature. They vary greatly in size and shape; in their habits and habitats; in their growth requirements; in their ability or lack of ability to produce disease, and in many other ways. Thousands of different bacteria have been isolated and studied, and new ones are constantly being discovered. Fortunately, out of this great

number, only a few are capable of causing disease, in fact many of them are of distinct benefit to mankind. An organism capable of producing infection is termed a *pathogen*.

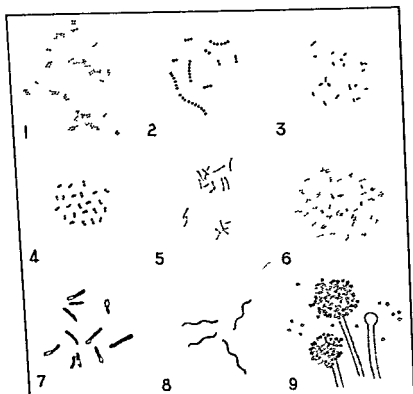


FIG. 2.—Various forms of microorganisms which are pathogenic for the fowl. 1 *Staphylococcus aureus* 2 *Streptococcus pyogenes* 3 *Salmonella pullorum* 4 *Pasteurella multocida* 5 *Mycobacterium avium* 6 *Brucella abortus*, 7 *Clostridium botulinum* 8 *Spirocheta gallinarum* 9 *Aspergillus fumigatus* (1 to 8 are $\times 1000$ 9 is $\times 300$) (Courtesy of Dr S. E. Park.)

As to form, bacteria occur in three principal shapes, *viz.*, rods (bacilli), spheres (cocci) and wavy threads (spirillae). In each of the three groups wide variations are commonly found, *e.g.*, the rods may be short, long, slender, plump, straight, curved, have rounded or square ends, possess motility or be unable to move. Some rods are also capable of producing

spores or "seeds" which are very resistant, and which account for the presence of certain infections on the same premises year after year. Under favorable conditions the spores develop into the usual or vegetative form of the micro-organism and are again highly infectious. The cocci vary greatly in size, usually appear round, and may occur singly, in pairs (diplococci), in chains (streptococci) or in masses (staphylococci). The spirillæ exhibit the same general variations as those noted in the rods. They may have but one curve in their body, in which case they are called vibrios, or they have a corkscrew appearance and possess many curves. To assume that a certain microorganism always retains the same form as well as other characteristics would be to ignore recent findings from which it has been learned that many organisms are capable of wide deviation from their usual type. Marked changes in the form of some organisms can be produced at will in the laboratory by varying the food, temperature or other factors concerned in their growth.

Viruses.—Viruses are disease-inducing agents which are capable of passing through laboratory filters fine enough to hold back the ordinary types of bacteria. Until recently, the only proof of the existence of viruses was their ability to reproduce disease upon introduction into the body of a susceptible animal. Development of the electron microscope with its greatly increased powers of magnification has made it possible for certain viruses to be observed, thus providing additional means for studying them. Viruses produce some of the most important diseases of poultry; *e.g.*, fowl pox, laryngotracheitis, and Newcastle disease.

Protozoa.—Protozoa are small one-celled organisms, the smallest and simplest in the animal kingdom. Out of the vast numbers of protozoa known at the present time, only a few are capable of inducing disease in poultry. They may produce disturbances in various parts of the body (coccidiosis in the intestines, blackhead in the liver and intestines). Although protozoan diseases in poultry are few in number they are economically among the most important.

Molds (Fungi).—Molds or fungi comprise a large group of the lower forms of the plant kingdom. They are abundant in

Nature and evidence of their growth is familiar to everyone. Their characteristics vary greatly as do those of the bacteria, and out of the vast number of molds known and classified, a few are capable of producing disease in susceptible hosts. There are three mold-caused diseases of particular interest to the poultryman, viz. favus, aspergillosis and thrush.

Much remains to be learned of the exact manner in which the various agents induce disease. Some for example while harmless in themselves are capable of producing toxins which under certain conditions are productive of disastrous results (*Clostridium botulinum*). Others are capable of actual invasion of an organ or part and multiplication therein, their activities resulting in disease (*Mycobacterium avium*). It is not necessary for an organ or part actually to be invaded in order to be affected by the presence of infection in the body. The products of infective processes often toxic in nature may be transported to any part of the body by the blood stream or the lymph stream and induce in that part a severe reaction. Finally, in many instances the exact manner in which certain infections produce such deleterious effects is not known and further research must be looked to for a solution of the problem.

CLASSIFICATION OF DISEASES

It is impossible to adopt any hard and fast rule in the grouping of diseases. Classification is usually based upon one or more characteristics of the condition and for our purpose diseases may be classified as follows, with one or more examples of each group being given in parentheses.

(a) As to cause

- 1 Bacterial—those caused by bacteria (tuberculosis, fowl cholera)
- 2 Virus—those induced by viruses (fowl pox, fowl pest, laryngotracheitis)
- 3 Protozoan—those for which protozoa are causative agents (blackhead, coccidiosis)
- 4 Parasitic—those caused by parasite infestation (ascariasis, teniasis)

5. Fungous—those caused by molds or fungi (aspergillosis, favus).
6. Unknown etiology—those for which the causative factor is as yet unknown (certain tumors).

(b) *As to infectiousness.*

1. Infectious—those caused by the entrance, multiplication and activities of any infectious agent (fowl typhoid, pullorum disease, fowl pox).
2. Non-infectious—those not induced by infection (gout, vitamin deficiencies).

(c) *As to transmissibility.*

1. Contagious—those diseases (also infectious) which are readily communicated to a susceptible individual (laryngotracheitis).
2. Non-contagious—those diseases not communicable to others (bumblefoot).

(d) *As to duration.*

1. Peracute—those which are excessively quick in their action (fowl plague).
2. Acute—those having a short course (fowl cholera).
3. Subacute—those slightly less active than acute (fowl pox).
4. Chronic—those which extend over a long period of time (tuberculosis).

(e) *As to occurrence.*

1. Sporadic—those not widely diffused; occurring here and there (fowl cholera).
2. Endemic—those confined to a district or locality (laryngotracheitis).
3. Epizootic—those prevalent over a wide area, affecting many individuals, and having a rapid spread (fowl plague).

From the foregoing outline it is obvious that in order for a certain disease to be classified, several characteristics must be taken into consideration. It should also be borne in mind that the manifestations and course of a particular ailment are not always the same—the condition may be acute in one instance and subacute or even chronic in others.

THE BODY DEFENSES

The body is constantly being exposed to infectious agents, and it is evident that were it not for definite means of resistance on the part of the body these pathogens would soon destroy the higher forms of life. The first line of defense is the skin, which in an unbroken healthy state offers resistance to infection to a remarkable degree. The lining membranes of the mouth, nostrils and other body openings, when uninjured, also resist infection.

Once an infectious agent has gained entrance to the body, however, there is normally an immediate response on the part of the defensive mechanism. If immune bodies or antibodies against the invading organism are present in sufficient quantity, they are called into action and the infection overcome. There are also present in the blood certain types of white blood corpuscles capable of destroying germs by ingesting them (phagocytosis). This function is so well defined that these white cells have been termed the "police-men" of the body.

If, on the other hand, the defensive forces of the body have been lowered by any means such as cold, starvation, injury, parasitism, malnutrition or the presence of other disease or if specific protective antibodies are not present, the resistance offered to the infectious agent is feeble and the invader is enabled to gain a foothold. Just as the body has the ability of organizing for defense, many of the infectious organisms have the ability to produce substances (aggressins) which paralyze the phagocytic cells and permit the bacteria to entrench themselves.

If the resistant forces of the body are capable of overcoming the infection, recovery ensues, if such forces are not equal to the task, the disease process goes on with the final outcome in doubt. It is obvious that there is constantly going on in nature a conflict mostly unobserved but upon the outcome of which depends the welfare of all higher animals.

METHODS OF DISEASE DISSEMINATION

The prevention of disease dissemination requires an understanding of the means by which the spread of infection takes

place. Numerous factors and agencies play a part in the spread of disease, whether from one individual to another in the same flock, or from one flock to another. Some of the facts pertaining to the dissemination of disease are therefore briefly discussed.

Infectious agents may escape from the body of an affected individual (a) in excreta, (b) in discharges from involved organs or parts and (c) by means of blood-sucking insects.

After leaving the body of the affected bird, the infection may be passed on to susceptible individuals in a number of ways.

(a) *By Means of Water* — Infectious discharges or parasite eggs which happen to fall in the watering trough or into a pool of water in the yard are readily picked up by susceptible fowls if care is not used to prevent such contamination. Adequate drainage of yards and runs is, therefore, essential.

(b) *By Means of Soil* — Under ordinary conditions soil offers a ready harbor for bacteria and parasite eggs, some of which are capable of surviving for long periods of time in this medium. Because of the habit of fowls of constantly picking up particles from the ground, the opportunity for them to pick up infectious material is ever present.

(c) *By Means of Air* — Although probably of not so much importance in the actual dissemination of disease as are soil and water, the air nevertheless is capable of transmitting infection from one bird to another. This is particularly true in the case of respiratory diseases, such as laryngotracheitis.

(d) *By Means of Contact* — Cohabitation of infected and susceptible fowls offers a ready means of spreading disease, not only by actual contact but also by the constant contamination of the soil and water to which the birds have access. Because of the ease of dissemination under such conditions isolation of all visibly affected birds should be carried out without delay.

(e) *By Means of Carriers* — Carriers are those fowls which, although they are not visibly affected, are nevertheless harboring the causative agent of a certain disease. This is particularly true in the case of birds which have undergone infection recovered and subsequently harbored the causative agent. It is difficult and at times impossible to detect such

fowls There is great danger of such undetected carriers serving as foci of infection from which the disease may spread to the susceptible members of the flock.

(f) *By Means of Vectors*—The term vector is applied to those creatures such as flies, mosquitoes, ticks, fleas, etc., which, without being infected themselves, are capable of transmitting infectious material from one fowl or animal to another. This is usually accomplished by the insects feeding in turn on infected and susceptible subjects.

(g) *By Mechanical Means*—Such transmission may take place by the hands, shoes and clothing of attendants, and by moving feed and watering troughs or other equipment from one place to another. Wild birds may also serve as disseminators of infection.

(h) *By Shipment of Infected Chicks or Fowls From One Place to Another*—It is by this means that the tremendous spread of certain infections, notably pullorum disease, commonly occurs. The exposure of healthy fowls while temporarily away from the flock at poultry shows or other exhibitions must also be considered.

From the foregoing it is readily seen that diverse and ever-ready means exist for the introduction and perpetuation of diseases and parasitoses in the flock. To maintain a healthy, vigorous and profitable flock the poultryman has no choice but to apply those principles of sanitation and disease prevention which are known to be sound, and to keep a constant lookout for the first appearance of trouble.

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Chapter 3

THE ANATOMY OF THE FOWL

SOME knowledge of normal structure is essential to an appreciation of the changes which result from disease, but exhaustive treatment of the subject is impossible in a book of this kind. The purpose of this chapter is to give a concise presentation of the facts about the structure of the fowl as related to normal functioning, which will perhaps be sufficient for most persons who have to deal with the fowl in health and disease. The reader who is looking for a detailed discussion of the finer points in anatomy and histology is referred to the books and articles listed at the close of the chapter.

The Fowl as a Living Organism — In comparison with most other animals, birds are often spoken of as fast-living creatures. Structurally, they are among the most highly specialized of vertebrates, many of their modifications being in the nature of adaptations for flight. Their coating of feathers is sufficient to set them apart from all other forms, but they are also characterized by being warm blooded, by having a high metabolic rate, and by the fact that development of the young takes place, for the most part, outside the body of the mother.

The body temperature of the fowl is higher than that of other domestic animals, and because of a more or less regular diurnal variation the recorded temperatures show a greater range than is found in other farm animals. The average noon temperature is 107.5°F , with a variation in the normal daylight temperature, regardless of breed, from 105° to 109.5°F . Average temperatures for other species of domestic poultry are approximately as follows: duck, 108° , goose, 105° , turkey, 106° , guinea, 107° , pigeon, 109°F .

The fowl is a rapid breather and its pulse-rate is high. For fowls at rest (on the roost at night) the normal number of respirations per minute is about 14-22, and the heart-rate is about 300. Any excitement will cause a rapid acceleration

in the heart-rate In the young chick the increase is quite remarkable Bogue (1932) observed that dropping a twenty-four-hour-old chick caused the heart-rate to increase from the normal 300 to as much as 560 beats per minute

Feathers—Feathers are epidermal structures, partly embedded in follicles of the skin, and varying greatly in size, color and shape They help to protect the bird from physical injury, and are of a great deal of aid in keeping the body warm The wing feathers are, of course, essential to flight

Most birds are entirely covered with feathers, but in only a very few species do feathers actually grow from the entire surface of the skin In most species, including the fowl, they are arranged in definite areas or feather tracts, called *pterylae*

Chandler (1916), who has made a very extensive study of the structure of feathers, recognizes three types—plumules, filoplumes and contour feathers The plumules are small, downy feathers which are usually completely covered by the contour feathers in adult birds The filoplumes are excessively slender and rather difficult to see They appear as hair-like structures and are never developed in sufficient numbers to be of any possible value as a covering They are really degenerate feathers in which the barbs, or side-branches, never become attached to the shaft, being "shed" as the shaft grows They are usually entirely covered by the contour feathers

Contour feathers, in their various forms and sizes, include practically all of the diverse kinds of plumage commonly seen on the body of a bird They include the remiges, or wing feathers, the rectrices, or main tail feathers, and the coverts, or exposed body feathers, as well as the less conspicuous ear-coverts, eyelashes and facial bristles which are found in some kinds of birds

The parts of a typical feather are the quill, or main stem, which includes the hollow basal portion, or calamus, and the shaft or rachis, the barbs, branching from the shaft, the barbules, branching from the barbs, and the barbicels, branching from the barbules The smooth apparently continuous appearance of the vanes of such feathers as the wing primaries is brought about by innumerable fine hooks

on the ventral barbs which serve to hold the parts of the feather solidly together. The intricate nature of feathers is apparent from the statement of Berbe (1906), who estimates that on a wing feather from a common pigeon there are about 1200 barbs and nearly 1 000 000 separate barbules.

It is worth noting that the large feathers of the wing and tail are definite in number, and that they are molted and replaced as a rule in a regular order. This fact is put to practical use in judging fowls for egg production, because it furnishes a basis for estimating the length of time that certain hens have been out of production.

The Skin—The skin of the fowl is comparatively thin, and consists of an outer layer, the epidermis, and an inner layer, the dermis. In the areas which are covered with feathers the epidermis is dry and covered with fine scales. These scales are constantly being shed and replaced as new skin tissue is formed.

There are no sweat glands in the skin of the fowl, nor are there any sebaceous glands except the uropygial or preen gland located at the base of the tail.

The scales on the shanks and feet, the toenails, the beak and the feathers are modifications of the epidermis, while the comb, wattles and earlobes are developments of the dermis.

The skin is sensitive to touch stimuli applied to the feathers. It is also supplied with numerous erector, depressor and retractor muscles by which the feathers are provided with a considerable degree of movement.

The Muscles—In the fowl, as in other animals, there are three kinds of muscles. Involuntary, or smooth muscles, are found in the walls of the alimentary canal, blood vessels and other tubular structures of the body. Voluntary, or striated muscles make up most of the edible portion of the fowl, and are of primary use in moving the various parts of the skeleton. The third kind is the heart or cardiac muscle which, of course, is involuntary but which has some of the characteristics of striated muscle and is therefore usually placed in a class by itself.

One fact about the muscular system of the bird which deserves special mention is the development of the large

muscles which control the major movements of the wing. The greater part of this muscle group appears to be on the body proper because of the extensive attachment to the sternum. Bradley (1938) states that the muscles in this region weigh about as much as do all the rest of the muscles together, and that they make up about one-twelfth of the weight of the entire body.

An interesting point in the musculature of the legs and feet has to do with the reflex nature of perching. The toes are automatically flexed when the intertarsal joint is bent. The fowl is therefore able to grasp the perch without effort, and is prevented from falling while asleep.

The diaphragm of the fowl is usually described as rudimentary, but McLeod and Wagers (1939) have shown that it consists of two parts. A very thin tendinous sheet, attached to the sternum, the 6th and 7th ribs, and the 6th thoracic vertebra, separates the thoracic and abdominal cavities. A second pulmonary diaphragm is present as a horizontal sheet dividing the thoracic cavity into an upper and lower section.

The Skeleton.—The skeleton of the fowl is compact, light in weight and very strong. Many of the long bones are hollow, which helps to make them light; and many of them are fused together, forming very strong structures to which the large flight muscles are attached.

It is customary to consider the skeleton of the fowl as consisting of axial and appendicular portions. The former includes the skull, vertebral column, ribs and sternum; while the latter includes the wings, legs and their supporting structures.

The vertebral column of the fowl differs from that of mammals in several particulars. That part which forms the skeleton of the neck is long and freely movable, but the balance of it is rigid, containing many fused bones. Some of the thoracic vertebræ are fused to form a firm base for the attachment of the wing and its muscles, and even more extensive fusion occurs in the lumbar and sacral regions. The hip bone is solidly fixed to the vertebral column, an arrangement which is necessary for strength, since the pelvic bones do not join ventrally as they do in mammals. This is an adaptation

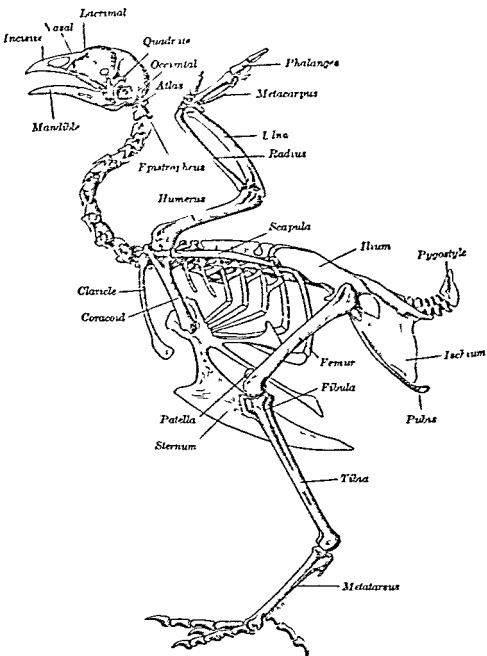


FIG 3 —The skeleton of a fowl.

of the pelvis to permit the laying of eggs of relatively large size. The pubic bones are thin, narrow strips with free posterior ends. This fact is of interest in connection with the physiological changes which take place in females as they go in and out of laying condition. The pubic bones are well spread in a laying hen.

The Respiratory System.—The respiratory system of fowls is quite different from that of mammals. A relatively long trachea connects the larynx with the syrinx, or lower larynx, where the trachea divides into the two bronchi. The voice of the fowl is produced in the syrinx. The air sacs are also necessary to voice production, for if the cervical air sacs are punctured no sounds come from the fowl.

The lungs are firmly attached to the thoracic wall and are deeply indented where they fit around the ribs. A bronchus passes through the entire length of each lung and connects finally with the corresponding abdominal air sac. This makes the air sacs important reservoirs of inspiration and expiration. McLeod and Wagers (1939) point out that there are no blind ends in the bronchial system. The air passages are continuous, with many anastomoses. It is probable that air moves more or less continuously through the system instead of in and out.

There are, in all, four pairs of air sacs ranging in position from the neck to the abdomen, with a single median sac located in the cavity of the thorax. They connect with the bronchi and with the cavities of many of the bones. That the connection with the bones is really substantial is shown by the statement of Beebe (1906) who says that a bird which has had its upper arm bone broken with shot is able to breathe through the splintered end of the bone when the windpipe is completely choked with blood.

The Circulatory System.—The heart lies just in front of, and partly between, the two lobes of the liver. It is conical in form and is surrounded by a thin, membranous sac known as the pericardium.

The single pulmonary artery arises from the right ventricle and divides into right and left branches, one going to each lung. The aorta leaves the left ventricle, gives off the right and left brachio-cephalic arteries, then turns to the rear and

passes along the vertebral column giving off paired and single arteries to supply the various organs and parts of the body

The pulmonary veins one from each lung, join just before reaching the left auricle of the heart. The blood from other parts of the body is brought to the right auricle by a single posterior and two anterior vena cavae. As in mammals, the blood from the stomach and intestines is carried to the liver by the portal vein.

Bradley (1915) states that the body of the fowl is richly furnished with lymph vessels, the largest being the right and left thoracic ducts. These run along the vertebral column and empty into the jugular veins. He also states that though lymph vessels are numerous, lymph glands are few in number and small in size.

The Blood of Fowls—The blood makes up about 5 per cent of the empty live weight of the fowl, being about 75 per cent water and 25 per cent solids. It consists of a straw-colored fluid portion, the plasma, and several types of corpuscles or blood cells. The plasma contains a substance called fibrinogen which is the precursor of fibrin. Fibrin is essential to normal clotting, as can be shown by whipping normal blood until the fibrin is removed. Defibrinated blood will not clot. When both the fibrin and the blood corpuscles are removed as in clotting, the remaining portion is called serum. The corpuscles are very small and are of two principal kinds, red and white.

The red blood cells (erythrocytes) are the most numerous of all the cells, ranging from 2,000,000 to 4,000,000 per cubic millimeter of normal fowl's blood. They are oval in shape and flattened and as seen individually under the microscope they are straw-colored. When seen in masses in the presence of oxygen they have a distinctly red color. The red cells of the fowl contain nuclei, differing in this respect from the red cells of most mammals. One of the important constituents of the red blood cells is hemoglobin, a protein substance having to do with the transportation of oxygen to the various parts of the body. The color of the blood depends upon the state of oxygenation of the hemoglobin.

The white blood cells (leukocytes) are, in general, larger than the red cells and are much fewer in number. Each

cubic millimeter of normal fowl's blood contains from 15,000 to 30,000 white cells. It is the leukocytes which play such an important part in the defense of the body against infections. They have the property of amoeboid movement and can actually leave the capillaries and enter other tissues of the body. Because of the ability of some of them to ingest and destroy invading organisms, they are sometimes called "phagocytes." These cells are divided into five principal groups on the basis of well-marked differences in size, structure and staining reactions.

The Nervous System.—The nervous system consists of two principal divisions or parts: the cerebrospinal, composed of the brain and spinal cord; and the sympathetic, made up of a series of nerve centers along the spinal cord. The brain is made up of the cerebrum, cerebellum and medulla oblongata, and the entire structure is covered by three meninges or membranes.

The cerebrum is the largest part of the brain and is divided into two hemispheres. It is interesting to note that the surface of the fowl's brain is smooth, in contrast to the convoluted surface in the brain of mammals. The cerebellum, or small brain, is ovoid in form and its surface is indented by numerous transverse ridges or folds. The medulla oblongata is that portion of the brain which connects the brain proper with the spinal cord. It is this part of the brain which must be pierced by the killing knife in order to loosen the feathers for dry picking.

The spinal cord is cylindrical, white in color, and extends from the medulla oblongata to the coccygeal portion of the vertebral column. Branches arising from it extend to various parts of the body.

The brachial plexus is a relatively large structure, composed of the last three cervical and first spinal nerves, and is located deeply in the region where the wing joins the body. The lumbo-sacral plexus is the group of nerve roots which fuse to form the sciatic nerve. The sciatic nerve is the large double nerve trunk which arises from the spinal cord by four or five heads dorsal to the kidney. It is buried in the muscles of the leg, and is readily found by cutting lengthwise through the inside thigh muscles.

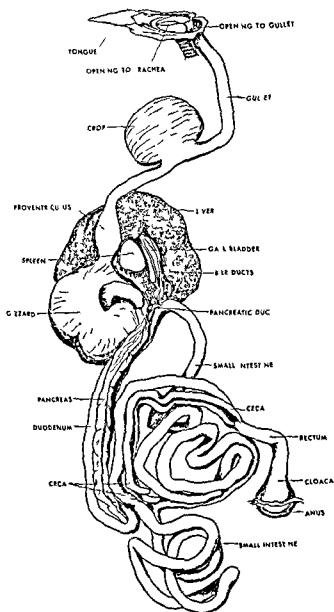


FIG 4 —The digestive system of the fowl (Courtesy of F B Adamstone)

The sympathetic system furnishes nerve branches to the various abdominal organs and blood-vessels.

The Digestive System.—The digestive system of the fowl includes the alimentary tract and the accessory organs. In some respects it differs materially from that of mammals.

Mouth Parts.—There are no teeth, but in their place the fowl has a horny beak which is used in breaking up much of its natural food. The distinctive character of the mouth is shown also by the absence of lips and cheeks. In water fowl the bill is broad, flat and pliable except at the edges.

The tongue in fowls and turkeys is shaped like the barbed head of an arrow with the point directed forward. The barb-like projections at the back of the tongue serve the purpose of forcing the food toward the entrance to the gullet when the tongue is moved back and forth. In water fowl the tongue is wider, softer and more flexible.

The hard palate, which forms the roof of the mouth, is divided by a long, narrow slit in the median line, which connects with the nasal cavities. Several transverse rows of horny papillæ, all with their points directed backward, are located on the surface of the hard palate.

Gullet and Crop.—The gullet, or esophagus, is distinguished by its great expansibility. Just before it enters the body cavity it enlarges to form a pouch called the crop or ingluvies, which furnishes temporary storage for the feed eaten, much as does the paunch of ruminants.

The Proventriculus.—Beyond the crop the gullet contracts until well within the body cavity, when it expands somewhat as it merges with the proventriculus or glandular stomach. On casual inspection the organ appears to be little more than a wider section of the gullet, but close examination shows that its walls are much thicker. Much of this thickness is accounted for by the presence of a layer of glands from which an acid gastric juice is secreted.

The Gizzard.—The gizzard, or muscular stomach, which is separated from the proventriculus simply by a constriction in the canal, is oval in form, flattened from side to side, and is composed of two pairs of red, thick, powerful muscles covered internally with a thick, horny epithelium. Beneath the ridged, leathery lining are glands from which the inner

surface layer is produced. In other words, the gizzard lining is really a hardened glandular secretion known as the "pseudo-cuticula." The opening into the duodenum is close to the one from the proventriculus and both are on the dorsal side of the gizzard.

A point of considerable interest is that the gizzard varies in condition according to the diet. Chickens fed exclusively on all mash rations have softer and less muscular gizzards than do those fed exclusively or largely on hard grains, especially if grit is not furnished.

The Intestine—The first section of the intestine is the duodenum. It is a folded loop, about 5 inches long in the mature fowl and encloses the pancreas. Its termination is marked by the pancreatic and bile ducts which pour their respective juices into the intestine at very nearly the same point.

There is no line of demarcation between the parts of the intestine corresponding to the mammalian jejunum and ileum, this portion of the intestine being arranged in folds and loops and supported by a thin membrane, the mesentery. Newton in his Dictionary of Birds, has called attention to the regularity of looping within a species, and to the similarity of looping in closely related species.

The large intestine in the fowl, corresponding to the mammalian colon and rectum is very short. At its junction with the small intestine, two blind pouches the ceca are attached. They are small in diameter at their junction with the intestine but enlarge somewhat toward their ends. They lie along the small intestine, with their closed ends forward.

The large intestine empties into the cloaca, a chamber common to the digestive and urogenital systems, which opens externally at the anus or vent. The cloaca is divided into three parts or sections, the coprodæum with which the large intestine is continuous, the urodæum, into which the ureters and genital ducts open and the proctodæum, from the dorsal wall of which an opening leads into the bursa of Fabricius. The bursa is of considerable size in fowls three and four months old but has nearly disappeared by the time they are one year of age. Its function is not understood.

The Liver and Pancreas—Closely associated with the diges-

tive canal, though not a part of the tract itself, are the liver and pancreas. The liver is the largest gland of the body, consists of two lobes, and is dark reddish-brown in color. The outer surface of the liver is convex and smooth, conforming to the shape of the abdominal wall. The inner, or visceral surface, is irregularly concave as if molded to fit around the adjacent organs.

The right lobe of the liver is, as a rule, the larger and on its visceral surface the gall-bladder is located. Two ducts carry the bile from the liver to the duodenum. The one from the left lobe appears as a simple tube, but the one from the right lobe is enlarged to form the gall-bladder. The two ducts enter the duodenum together.

There is a progressive change in the color of the liver in young chicks during the third and fourth weeks after hatching, coincident with the disappearance of fat from the organ.

The pancreas is a long, thin, light-colored organ which occupies the space formed by the loop of the duodenum. The pancreas secretes an important digestive fluid, known as the pancreatic juice, which is carried to the intestine by three ducts. They enter the duodenum slightly anterior to the bile ducts. The pancreas is important also as the source of an internal secretion produced by the islets of Langerhans which occur throughout the organ.

Microscopic Anatomy of the Digestive Tract.—A very careful study of the microscopic anatomy of the digestive tract of the chicken has been made by Calhoun (1933). The earlier literature on the subject is reviewed, and an extensive bibliography is included. Thirty-nine plates, most of them photomicrographs, are given. The reader who is interested in the histology of the digestive tract of the fowl will find it profitable to examine her excellent report.

Rosenberg (1941) has described in detail the micro-anatomy of the duodenum of the turkey. For a still more complete study of the histology of the intestine of various kinds of birds, see the series in German by Clara (1925, 1926, 1927).

The Urinary System.—The urinary organs of the fowl consist of the kidneys and ureters. The brownish, highly vascular kidneys are attached to the vertebral column just

behind the lungs. They are elongated organs and are lobed to fit the depressions in the fused bones of the hip region. A single ureter connects each kidney with the cloaca. The urine is discharged into the cloaca, which serves as a urinary bladder. Hester, Essex and Mann (1940) have described the

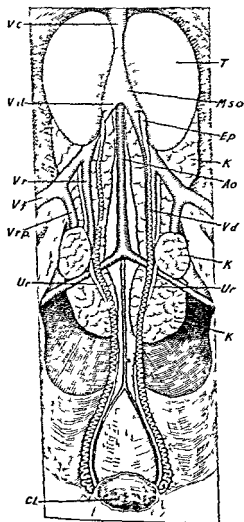


FIG 5.—The reproductive and urinary organs of the male fowl. T, testis; Vd., vas deferens; K, kidney; Ur, ureter; Cl., cloaca. (Courtesy of L. V. Domm.)

normal urine of fowls as a cream-colored, thick, tenacious, mucoid material containing an abundance of urates which readily settle out forming a semi-solid mass. The largest volume which they obtained in twenty-four hours was 123.5 cc. They found that the disturbance of moving and preparing fowls for observation was sufficient to cause considerable diuresis, which apparently explains the large urine flow reported by some investigators.

The Reproductive System of the Male — The organs of reproduction in the male fowl consist of the two testes, each with its vas deferens leading to the cloaca. The testes are attached to the dorsal wall of the abdominal cavity, near the anterior ends of the kidneys. In the mature male they are more or less ellipsoid, and light yellow in color, often having a reddish cast from the numerous blood-vessels on their surfaces. The testes are not necessarily of equal size, and both are normally larger during the breeding season than at other times of the year as, for example, when a male is molting.

The inner, or medial surface of each testis is slightly concave, and on it appears a flattened projection which corresponds to the epididymis of mammals. From this the slender, wavy vas deferens begins. This duct runs beside the ureter on its way to the cloaca, into which it opens through a small papilla. The so-called rudimentary copulatory organ has no connection with either vas deferens.

The three parts of the cloaca previously mentioned are separated by transverse folds. In the median ventral portion of the fold between the urodæum and the proctodæum of male chicks is a very small, shiny or glistening projection which is variously referred to as the genital eminence, male process, or rudimentary copulatory organ. It is by everting the cloaca to expose this rudimentary organ that day-old chicks are sorted into pullets and cockerels.

The Reproductive System of the Female — The organs of reproduction in the female fowl are the ovary and oviduct. In early embryonic life there are two gonads in the female, as well as in the male, but normally only the left one develops, the right being found, if at all, only as a functionless rudiment. A few cases have been reported in which both a right

and left ovary and oviduct were present and functioning, but these are rare.

The left ovary is attached to the dorsal wall close to the left kidney. When in functional condition it appears as a

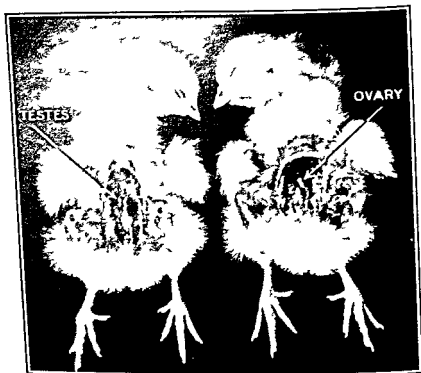


FIG. 6.—The location and appearance of the internal sex organs of baby chicks

LEGEND FOR FIG. 7

FIG. 7.—The reproductive system of the female fowl. 1 The ovary. 2 a partly developed ovum. 3 still larger ova—the lower one is nearly ready to leave the ovary. 4 the stigma—a region in which there are normally no blood-vessels. 5 an empty follicle from which the yolk has entered the oviduct. 6 lip or margin of the funnel. 7 opening or mouth of the funnel—when an ovum is about to leave the ovary it is normally enclosed by the edges of the funnel and guided into the oviduct. 8 a yolk which has just entered the oviduct. 9 albumen-secreting portion of the oviduct. 10 albumen which is secreted around the yolk. 11 yolk. 12 the germinal disc. 13 anterior end of the isthmus in which the shell membranes are formed. 14 the uterus or shell gland. 15 the large intestine. 16 the abdominal wall cut and laid back. 17 anus or vent. (Partly diagrammatic.) (After Duval.)

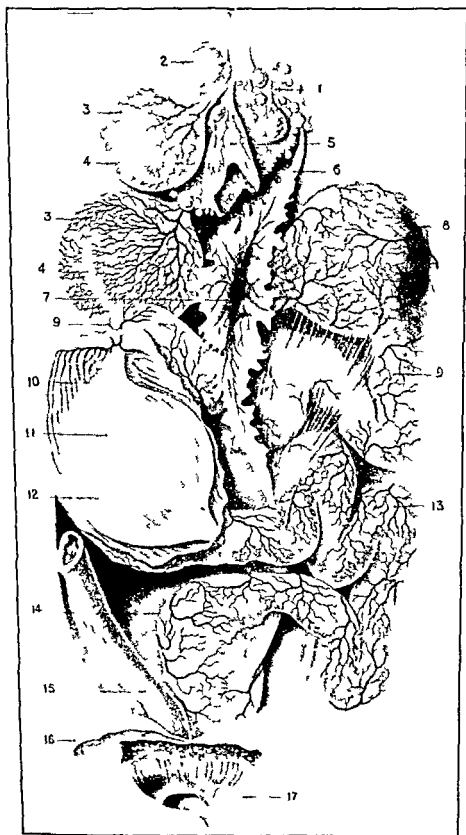


FIG 7

capable of producing marked effects upon the physiological processes of the body, and the glands which produce them are known as endocrine glands. Their functions are far from being completely understood, though much information concerning them has become available during recent years. They are mentioned here in order to call attention to their size and location in the body.

The list of endocrine glands in the body of the fowl includes the adrenal glands, the gonads, the pineal gland, the pituitary body, the thymus, the thyroids and the parathyroids.

The adrenal glands, or suprarenal capsules, are small, irregularly oval bodies lying adjacent to the testicles or ovary and near the anterior lobe of the kidneys.

The pineal gland is a very small body found in a median position in the brain, just behind the two cerebral hemispheres and in front of the cerebellum.

The pituitary body, or hypophysis, is a small rounded body found beneath the brain and attached to it by a slender hollow stalk. It is located just behind the point of origin of the optic nerves.

The paired thymus glands are prominent in young birds. They extend along the entire length of the neck, reaching maximum size at about four months of age, or just before the fowl becomes sexually mature. They then begin to diminish in size and may be entirely absent in old birds.

The two thyroids are small, oval, reddish glands found near the base of the neck and located close to the jugular veins.

The parathyroids are very small bodies and are found in pairs near each thyroid.

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as four hours. The required dosage was about 0.1 gm. per pound of body weight.

Local anesthetics are seldom used in poultry practice, one notable exception being when creolin is to be put into the eyes for the destruction of eye worms. Butyn is a very satisfactory anesthetic for this purpose. Sweebe (1925) states that a 2 per cent solution of butyn is as effective as a 5 per cent solution of cocaine, and that it is cheaper.

ABDOMINAL SURGERY

Opening of the abdominal cavity is necessary for at least four operations on fowls. These are caponizing, cecal ablation and relief from either ascites or the accumulation of yolk material in the body cavity.



FIG. 8

FIG. 9

FIG. 8.—Young cockerels ready to caponize.¹

FIG. 9.—A handy board for holding the bird during the operation.

Caponizing.—The castration of male fowls is commonly referred to as caponizing. The operation should be performed when the young cockerels weigh about $1\frac{1}{4}$ to $1\frac{1}{2}$ pounds each. This will be when they are from five to ten weeks of age, varying somewhat with the breed and with the conditions under which the chickens have been raised. Those to be operated on should be held without food for eighteen to twenty-four hours in order that the intestines

¹ FIGS. 8 TO 19 FROM Waite's "Poultry Science and Practice," published by the McGraw-Hill Book Company.

Chapter 4

POULTRY SURGERY

BECAUSE of the small value of the individual fowl, surgery is not often used as a means of treating or removing pathological conditions. It is nevertheless true that fowls make excellent surgical subjects because of their marked resistance to pyogenic, or pus-forming bacteria. As evidence of this it is merely necessary to cite the practice of caponizing, which can be successfully carried out with no more regard for the prevention of infection than the exercise of ordinary cleanliness. In none of the larger farm animals would it be possible to open the abdominal cavity in such a manner without great danger of infection.

Anesthesia — Because of the peculiar structure of the respiratory system in fowls, the common general anesthetics are not satisfactory for use by inhalation. Fatal results often follow the use of ether or chloroform.

Some of the newer anesthetics, which can be given by intravenous injection, have greatly simplified the problem of anesthetizing fowls so that there is no longer any valid reason for avoiding anesthesia, at least for experimental operations.

Pento-barbital sodium (nembutal) has been found to be very satisfactory. Injection of 0.5 to 0.75 cc. in the wing vein will bring about immediate anesthesia, lasting for as long as two hours. Slow injection is recommended. Additional 0.25 cc. doses may be given as necessary, if a fowl is to be kept under anesthesia for several hours.

Durant and McDougale (1935) found that for complete anesthesia of turkeys it was desirable to use 1.1 cc. of nembutal for each 5 pounds of live weight.

Lee (1953) found chloral hydrate a safe and satisfactory anesthetic when administered by mouth in a 1 or 2 per cent solution. Although twenty-five to thirty minutes time was required for complete anesthesia, the duration was as long

as four hours. The required dosage was about 0.1 gm. per pound of body weight.

Local anesthetics are seldom used in poultry practice, one notable exception being when creolin is to be put into the eyes for the destruction of eye worms. Butyn is a very satisfactory anesthetic for this purpose. Sweebe (1925) states that a 2 per cent solution of butyn is as effective as a 5 per cent solution of cocaine, and that it is cheaper.

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¹ Figs 8 to 10 from Waite's "Poultry Science and Practice" published by the McGraw Hill Book Company

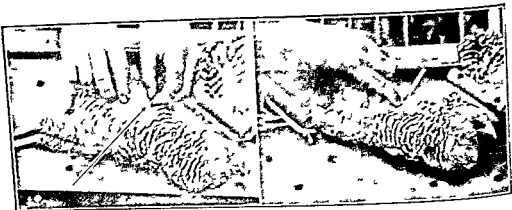


FIG 10

FIG 11

FIG 10 — Pulling a few feathers at the site of the incision

FIG 11 — Drawing the skin to one side preparatory to making the incision

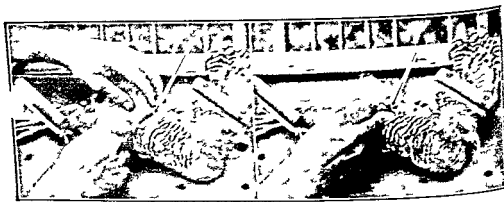


FIG 12

FIG 13

FIG 12 — The incision is made between the last two ribs.

FIG 13 — A spreader is inserted and the incision enlarged



FIG 14

FIG 15

FIG. 14.—Tearing the abdominal membranes to expose the testicle

FIG. 15.—A close-up view of the incision and the testicle

may become empty and fall away from the site of the operation. It is also well to withhold water during the last twelve hours, or even longer, as an abundance of water in the body tissues seems to increase the danger of hemorrhage.



FIG 16

FIG 16 —Removing the testicle.

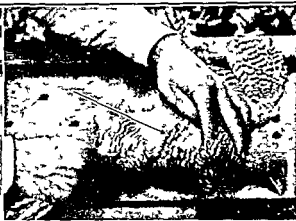


FIG 17

FIG 17 —When the spreader is removed the skin slips forward to cover the incision

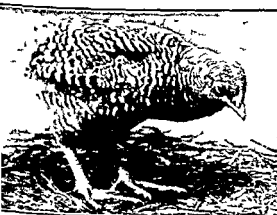


FIG 18

FIG 18 —Released after the operation—looking for feed

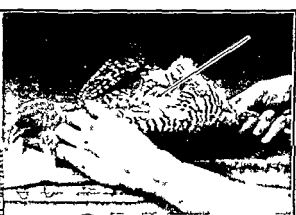


FIG 19

FIG 19 —After ten days—completely healed

The equipment needed for caponizing is rather simple. Some sort of work table of convenient height, and a means of restraining the fowls are the first requirements. The instruments needed consist of a sharp knife or scalpel, a

may become empty and fall away from the site of the operation. It is also well to withhold water during the last twelve hours, or even longer, as an abundance of water in the body tissues seems to increase the danger of hemorrhage.



FIG. 16

FIG. 16.—Removing the testicle.



FIG. 17

FIG. 17.—When the spreader is removed the skin slips forward to cover the incision.

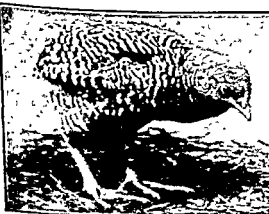


FIG. 18

FIG. 18.—Released after the operation—looking for feed.

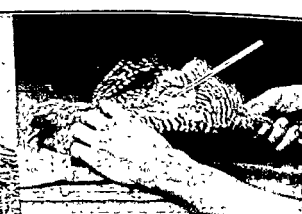


FIG. 19

FIG. 19.—After ten days—completely healed.

The equipment needed for caaponizing is rather simple. Some sort of work table of convenient height, and a means of restraining the fowls are the first requirements. The instruments needed consist of a sharp knife or scalpel, a

testicle "from below." This is especially helpful when both testicles are to be removed from one side.

After the young cockerel is in position and fastened securely, pluck a few feathers from the immediate area through which the incision is to be made. Using a bit of absorbent cotton, sponge the exposed skin with cold water and moisten the surrounding feathers so that they will lie down out of the way. From this point on, the operation should be performed as quickly as possible in order to cause a minimum of discomfort for the patient.

Find the last two ribs by palpation; slide the skin toward the thigh, making sure that the underlying thigh muscles are out of the way; force the knife through the skin and flesh between the ribs (some operators prefer to use the point, others the edge of the knife in starting the incision); lengthen the incision until it is about an inch long, keeping it in the center of the space between the two ribs and not too near the back; and insert the spreader in such a manner as to hold the ribs about $\frac{1}{2}$ inch apart.

This will bring the intestines into view beneath the peritoneal membrane and the abdominal air sac. With the hook tear through these membranes, if the knife has not already severed them. If the bird has been properly starved so that the intestines are well out of the way, the upper testicle should then be visible, attached to the dorsal wall of the abdominal cavity. It is normally yellow in color, but is sometimes rather dark; it varies in size, depending on the development of the bird, from that of a plump grain of wheat to that of a small bean.

If both testicles are to be removed from the one side, the lower one should be removed first so that any hemorrhage which may result will not interfere with the rest of the operation. The lower testicle is not usually visible through the incision and must be lifted into view with the forceps before it can be grasped. This part of the operation can be greatly facilitated by extending the incision around the bend in the ribs.

The delicate part of the operation is the actual removal of the testicle, because the entire organ and the connecting portion of the spermatic duct must be taken out in order to

tised because the operation is more difficult, and because there is no economic advantage to be gained by it

Elley (1913) has given an excellent description of the somewhat different technique which is followed in the castration of ostriches

Cecal Abligation—This refers to an operative technique developed in the course of experiments which were aimed at the control of blackhead in turkeys by surgical means. The surgical features and detailed technique have been described for fowls by Durant (1926), and for turkeys, with special reference to blackhead, by Durant (1930)

The general procedure is exactly the same as for caponizing, the incision being made on the left side between the last two ribs. The point of junction of the two ceca with the intestine is located opposite this point, and the parts to be operated on are easily lifted through the incision to a convenient position. If carefully done, only a very few small blood-vessels will be ruptured and but little hemorrhage will occur

Each cecum is tied off with two ligatures of catgut about 4 mm (not quite $\frac{3}{16}$ inch) apart, care being taken to draw the ligatures tight enough to close the passage into the main gut, yet not tight enough to cut through the outer cecal covering. The size of the suture material will depend somewhat upon the size of the bird, No. 4 being suitable for mature turkeys, and No. 3 for mature fowls

After the operation is complete, the organs are gently replaced and the wound closed by a single ligature. Durant states that if the ligature is passed around the two adjacent ribs and drawn tight, the wound is effectually closed and the development of wind puffs is practically eliminated. No special after-care is required

A point of special interest is that the ceca do not become necrotic, because their blood supply is not cut off. They gradually shrink from disuse and eventually become severed from the main gut, remaining suspended by their mesenteric ligaments as free-hanging sealed pouches. Some trouble is occasionally experienced in cases in which one or both ceca become greatly enlarged and distended in from seven to thirty-two months after the operation. Since the average

age at which this trouble develops in turkeys is slightly above eighteen months (Durant, 1930), it is a problem in the case of breeding turkeys but not in those being raised for market purposes.

Delaplane and Stuart (1933) report that the use of aluminum clamps in place of ligatures reduced the mortality from the operation to an average of 15 per cent, but did not entirely prevent the ceca from resuming a functional condition.

Schlotthauer, Essex and Mann (1933) used a different technique and reported still better success. They operated through an abdominal incision, about $1\frac{1}{4}$ inches long, parallel to the left pubic bone. The ceca were gently but completely crushed with a small hemostat before tying them off. Two ligatures were securely tied in place, one above and one below the crushed portion, before removing the clamp. These authors state that the operation was best performed on poults four weeks of age, and that such operated poults were effectively protected against blackhead.

Ascites—This is an excessive accumulation of serous fluid in the abdominal cavity. It is also known as abdominal dropsy. Treatment is rarely profitable, but may be worth while in the case of valuable birds. The usual treatment is that of Hill (1884), who reported immediate relief from the condition by tapping the abdomen with a small trocar and cannula. He removed 28 ounces of a greenish-colored, odorless fluid from a single fowl. Kaupp (1933) reports a typical case in which the abdominal cavity refilled, after draining, at the rate of about 12 cc. a day.

Eggs in the Abdomen.—It may sometimes be desirable, in the case of a valuable fowl, to operate for the removal of eggs or egg yolks which have accumulated in the abdominal cavity. McKenney (1929) reports a successful operation of this sort in which 4 large eggs and parts of 2 broken eggs were removed from a Leghorn pullet. The larger egg-yolks in the ovary were removed at the same time in order to permit the parts to heal before any further egg-laying could occur. The pullet made a complete recovery and began laying forty-six days after the operation.

Other Abdominal Operations—In connection with physiological studies of the processes of digestion and reproduction in the fowl it is frequently necessary to perform surgical operations which involve opening the abdominal cavity. Deutectomy, gizzardectomy, splenectomy, and resection of the oviduct are examples.

Deutectomy is a term which has been used to designate the operative removal of the yolk sac from newly hatched chicks. The usual purpose would be to obtain chicks with a minimal initial supply of vitamins or other nutrients in order to provide uniform lots of chicks for certain experimental studies in nutrition. The operation involves making an abdominal incision, manipulative extrusion of the yolk sac, cutting and searing of the yolk stalk with hot scissors, and suturing of the incision. The details have been described by Sloan (1936), and by Harvey Parrish and Sanford (1955).

A technique for the surgical removal of the gizzard has been described in detail by Burrows (1936). This procedure was used in studying the function of the gizzard by comparing normal and gizzardectomized fowls with respect to their ability to digest ground and unground feeds.

Rothchild (1947) has given a good description of the technique used in connecting the rectum to an artificial anus in the fowl so that urine and feces can be collected separately.

In a similar manner the physiology of egg formation is being studied by surgical removal of various sections of the oviduct and noting the manner in which eggs produced by the experimental hens differ from those laid by unoperated hens.

MINOR SURGICAL OPERATIONS

Surgical operations of a minor nature which may be performed on fowls include occasional treatment of wounds, operative relief for cases of crop-bound, dubbing of combs, cropping of wattles, and the removal of spurs or treatment to prevent their development.

Wounds—Wounds of a minor nature usually require no treatment of any sort, but occasional severe injuries will need attention if the fowl is of sufficient value to warrant

Cropectomy.—Fisher and Weiss (1956) studied the effect of surgical removal of the crop in young chicks on subsequent growth and feed consumption. Cropectomy proved to be a rapid and simple procedure with uncomplicated post-surgical recovery. A one and one-half inch longitudinal incision was made in the skin overlying the crop, and the crop was pulled through with forceps until the constricted area marking the junction with the esophagus was visible. A stout ligature was tied around the constricted region with No. 18 linen suture, and the crop severed just distal to the tie. The skin incision was closed with one No. 14 wound clip and the chicks returned immediately to a normal battery environment. The operation was easily performed on week-old chicks. Neither anesthetic nor aseptic technique was required, but sufficient prestarvation to empty the crop was desirable.

Impaction of the Crop.—This is often called "crop-bound," and consists of an excessive accumulation of coarse or stringy material in the crop. The opening to the lower esophagus may become obstructed, or the walls of the crop may become paralyzed by the constant distention and the food remains in the crop until it becomes sour.

If the impacted mass is not too large and firm it may often be removed by first introducing water into the crop through the mouth and then gently massaging the crop in order to work the mass out through the esophagus a little at a time. This is more easily done if the fowl is suspended head downward.

Surgical treatment is often necessary before the material can be removed. It is accomplished by making an incision through the skin and crop wall, and removing the accumulated mass a little at a time through the opening thus made. The incision in the wall of the crop should be not more than 1 inch long, and it should be made on the upper front surface of the crop, so as to be on the upper side when the fowl is standing. After cleaning out the food mass, the crop should be washed out with clean warm water, and the incisions in the crop wall and skin closed separately by placing several sutures in each. These may be made with coarse, white silk thread, or even with cotton thread. Each stitch should be made and tied separately, using knots that will not slip. It

is best to offer no food or water for about twelve hours, and to give nothing but soft food for a few days thereafter.

Most cases of impaction caused by fresh cut grass can be avoided if only short chippings of young, tender grass are used. Grass that has been cut 5 or 6 inches long is potentially dangerous.

Amputation of Comb or Wattles—In the treatment of edema of the wattles, or of frozen combs and wattles, or in the prevention of these troubles, it is frequently desirable to amputate the head appendages. Amputation of the comb is commonly referred to as "dubbing," and that of the wattles is sometimes spoken of as "cropping."

A razor blade or sharp scissors may be used. Hemorrhage following removal of the comb can often be much reduced by laying a clean feather over the cut surface. This helps to bring about clotting of the blood. Some poultrymen who dub many cockerels every year arrest hemorrhage by applying a small handful of flour or of whatever mixture of ground feed is at hand. Such methods of controlling hemorrhage are obviously not in accord with good surgical technique, but the pronounced resistance of the fowl to pyogenic organisms enables them to be employed with little danger of infection.

Healthy Leghorn cockerels may be dubbed successfully at almost any time up to six months of age. Surgical shears or common tinnerns' shears may be used, or the comb may be laid on a block of wood and cut off with a sharp knife. The operation should be performed on a mild day. Zero or near zero weather increases the likelihood of severe hemorrhage.

When dubbing of the comb is to be followed as a routine procedure with such large-combed fowls as Leghorns, one of the best methods is to decomb them as baby chicks. A fine pair of sharp scissors is convenient for the purpose. If the cutting is carefully done the chicks seem to suffer no pain and bleeding does not occur to any extent.

Trimming the Spurs.—One of the minor but often important surgical operations where breeding flocks are concerned is that of trimming the spurs of male birds to prevent laceration of the backs of the females. A small hack saw or a

sharp knife may be used, the object being to trim the spurs to a smooth, blunt end.

It is entirely practical, as shown by Smith (1932), to treat the spurs on young cockerels in order to prevent their development. The method consists in cutting off the spur cap close to the leg and rubbing the wound lightly with a stick of potassium hydroxide (caustic potash). The proper age for treatment is ten to twelve weeks for Leghorns, and fourteen to sixteen weeks for the heavier breeds, or before the spur cap is more than $\frac{1}{4}$ inch long.

Fractures.—Broken bones, especially the long bones of the legs and wings, heal readily if properly treated, but unless a valuable breeding bird is affected the best procedure is to butcher the fowl. If the bird is worth saving, it should be taken to a qualified veterinarian for treatment.

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Chapter 5

DIAGNOSTIC METHODS

THE fact that all is not well with his flock may be impressed upon the poultryman in any one of several ways. His fowls may start down in production; some of them may be visibly ill; or several dead fowls may be found without any previous warning as to the presence of illness among them. In any case no time should be lost in determining, if possible, the nature of the trouble and in instituting measures for the prevention of its spread to the remainder of the birds. It is not always possible to determine immediately the exact nature of an ailment, but many of the diseases of poultry produce changes which are apparent upon close examination. Much time can be saved and the findings more readily interpreted if the examination of a sick or dead bird is carried out in a systematic manner. Very few diseases produce symptoms that can be considered diagnostic, and for many conditions the symptoms displayed by an affected fowl are so similar as to be useless from the standpoint of differential diagnosis.

It is always well to examine the live bird for droopiness, lameness, discoloration of comb or face, or any unusual symptom which can be seen only in the live specimen, but detailed examination of the various parts can much better be carried out by postmortem examination. The relatively small value of the individual permits the sacrifice of several fowls for autopsy, if necessary.

POSTMORTEM EXAMINATION

The first requisites for a thorough and satisfactory post-mortem examination of a fowl are good light and a set of appropriate instruments. Only a few instruments are needed, but they should be sharp and clean. A pair of bone forceps or heavy serviceable scissors, a sharp-pointed butcher knife, a pair of small scissors having at least one pointed jaw, and a flat metal pan, which will prevent the escape of fluids and

which can be easily cleaned and disinfected after use, comprise all the equipment necessary for conducting a fowl autopsy.

If the bird is to be killed, this may be accomplished quickly and easily by breaking the neck. To do this, the legs and wing tips are grasped with one hand, and the head with the other hand in such a manner that it rests firmly in the hollow between the thumb and first finger. Bend the



FIG. 21 —Killing a fowl for autopsy by breaking the neck

head back at a right angle with the neck and push downward firmly until the bones in the neck separate. As soon as the neck is broken the operation should cease, as it is undesirable to pull the head off the neck. If the procedure is properly carried out, all hemorrhage from the ruptured blood-vessels in the neck will collect under the skin in the region of the break and thus be prevented from spilling.

As soon as the fowl has ceased struggling, it is placed back downward in the pan, or upon a table over which have

been spread several thicknesses of paper. The feathers should be thoroughly moistened, preferably with plain water, since most of the common disinfectants form an undesirable slime upon coming in contact with body tissues and fluids. The carcass should be opened without disturbing the internal organs, and with a little care this may be readily accomplished. Cut through the loose skin on each side between the body and the leg, then cut across just back of the end of the keel bone and connect the first two incisions. Push



FIG 22 —Postmortem examination The breast has been cut away to expose the internal organs.

the legs downward until they are dislocated at their connection with the body—this helps to prevent the carcass from rolling during the remainder of the examination. The skin is next torn from over the entire field by grasping the loose flap made by the first incisions and tearing forward toward the head. The heavy breast muscles are next cut through, down to the bone. Starting then at a point just back of the end of the breast bone, and using the heavy scissors, cut forward through the ribs on each side, and on through the coracoid and clavicle bones. The entire breast wall may

now be lifted upward and forward, bringing into view most of the internal organs

The presence of fluid in the abdominal cavity (ascites) is suggestive of peritonitis or a circulatory disturbance, yolk material in the cavity or spread over the organs points to ruptured yolks, and the presence of white, chalk-like flakes is indicative of visceral gout. An enlarged liver is suggestive of fowl typhoid (especially if the organ has a greenish sheen), visceral lymphomatosis or blackhead. White or grayish areas in the liver varying in size and consistency are noted in tuberculosis, blackhead, fowl typhoid and occasionally in coccidiosis.

The alimentary canal from the crop backward is now examined. An enlarged crop, full of sour stinking food or grass stems is diagnostic of impaction of that organ. The contents should be carefully examined for the presence of unusual material and the condition of the lining noted. The presence of a slimy, stinking, whitish, mucoid material over the crop lining is suggestive of parasite (capillaria) infestation or of mycotic infection. If the proventriculus is enlarged and the glands swollen and inflamed, these changes may be due to parasites (flukes, stomach worms) or an inflamed lining may suggest the ingestion of caustic substances. The gizzard is next opened and examined for the presence of small worms (gizzard worms) in the walls and under the horny lining.

The intestines are now opened over their entire length by means of the small scissors, and examined for inflammation (coccidiosis, worm infestation, fowl cholera), nodules (tuberculosis, lymphomatosis, tumors, tapeworms) and white spots (coccidiosis). Most of the roundworms and tapeworms are readily visible with the unaided eye, but a few require exceedingly close scrutiny for detection. If a portion of the bowel is submerged in clean water, some of the less conspicuous tapeworms may be seen attached to the intestinal wall. Magnification is necessary in some cases. At the junction of the large and small intestine are the two ceca, which may be enlarged, walls thickened and filled with bloody exudate (coccidiosis) or if the walls are thickened and a cheesy yellowish exudate present in the lumen, black-

head may be suspected. The small cecal worm is usually found only in the ceca.

Occasionally an egg is found obstructing the cloaca (egg bound). Intense inflammation of the vent accompanied by an extremely offensive odor is suggestive of cloacitis. Protrusion of the oviduct is evidenced by a protrusion of that organ through the vent. The spleen may be enlarged and mottled with gray areas (lymphomatosis) or it may be enlarged and contain hard, grayish, raised nodules (tuberculosis). Enlarged kidneys may be seen in fowl typhoid. The ureters may be distended with urates (vitamin A deficiency). Dried, shrunken, discolored ova are characteristic of pullorum disease, and occasionally of chronic fowl typhoid. The testes of males rarely show any changes.

The pericardial sac enclosing the heart may be thickened and contain an unusual amount of fluid (pullorum disease) or it may be covered with white, chalk-like flakes (visceral gout). The heart wall may show grayish firm enlargements (fowl typhoid, lymphomatosis) or be studded with small hemorrhages (fowl cholera).

The lungs may be pneumonic, dark and congested (fowl cholera), or contain firm, gray areas (lymphomatosis). In baby chicks the presence of small white nodules is very suggestive of pullorum disease, or if the nodules are black or greenish, aspergillosis may be present.

The eyes may be inflamed (colds, roup, eye worms) or contain cheesy exudate (white in vitamin A deficiency, yellowish in roup and fowl pox). Grayish discoloration of the iris is suggestive of lymphomatosis. Raised, rough, attached, yellowish, membranous patches in the mouth suggest the diphtheritic form of fowl pox. Round, raised, whitish, pin-point patches in the mouth and esophagus are characteristic of vitamin A deficiency. Swelling and edema of the larynx especially if accompanied by the presence of cheesy, yellow exudate, or blood-stained mucus in the trachea, are suggestive of laryngotracheitis.

The larger nerves, especially the brachial plexus of the wing and the sciatic nerve of the leg, should be examined for evidence of swelling and a change in color from the normal white to a gray, translucent appearance (neural type of

now be lifted upward and forward bringing into view most of the internal organs

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fully packed, and forwarded without delay. Upon completion of the autopsy, the carcass should be disposed of by burning, and all instruments, pans and equipment used in the examination thoroughly disinfected by soaking in some reliable disinfectant. (See Appendix, page 394.)

Preparation of Specimens for Shipment.—Typically affected live birds are far more satisfactory as specimens from which to make a diagnosis than are dead birds or organs from them. Postmortem changes occur rapidly after death and these may be so extensive in a short time as to preclude reliable findings. Bacteriological examination of birds dead for any length of time is usually unsatisfactory because of the rapid growth of contaminating microorganisms. It may be necessary at times, however, to submit a dead bird, and in that event the carcass should not be opened, but should be wrapped in several thicknesses of paper, carefully packed in a metal or strong cardboard box, plainly labeled both as to sender and destination, and forwarded so as to reach the laboratory in the shortest possible time. The submission of organs for bacteriological examination is not to be encouraged but tumors and parasites, or the organs suspected of containing either may be submitted by placing them in a 10 per cent solution of formalin in a small, fluid-tight container.

If live specimens are to be submitted, at least two typically affected fowls should be sent. It is not uncommon to find entirely different conditions in two fowls from the same flock, and presumably affected with the same disease. The birds should be crated to conform in detail with the regulations of the transportation agency, and sent so as to insure the earliest possible delivery to their destination.

Just as important as the specimens themselves is a complete history of the flock. This should include the number of fowls, number and age of fowls affected, symptoms shown, rate of spread and any other pertinent details.

LABORATORY PROCEDURES

It would be impractical because of space limitations, as well as wearisome to the reader, to include here a discussion of all the procedures which may be resorted to in the labora-

avian leukosis complex) The muscles should be observed for tumors or for the presence of small, maggot-like parasites (sarcocystis)

Diseases are rarely constant in their manifestations and it must not be assumed from the foregoing examples that the pathological changes mentioned are the only ones which

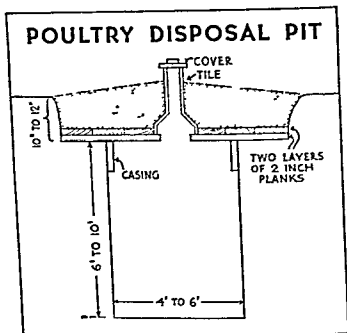


FIG. 23.—A deep covered pit such as the one shown here simplifies the problem of disposing of dead fowls (*Courtesy of Everybody's Poultry Magazine*)

may occur in a specific organ, or that such alterations always accompany the disease in question. The examples given are only suggestive of some of the more common findings, and the list is by no means complete either as to diseases or pathological alterations.

If it is desired to forward specimens of organs to a central laboratory for diagnosis, the part should be removed, care-

ature is maintained at about that of the body. The time for the appearance of growth varies greatly, but in most cases forty-eight hours suffices. If growth is present at that time the "colonies" (growth groups) are examined and careful observation made of such characteristics as number and size of colonies, consistency, color and shape. If a fluid medium has been used, the amount of growth, sediment,

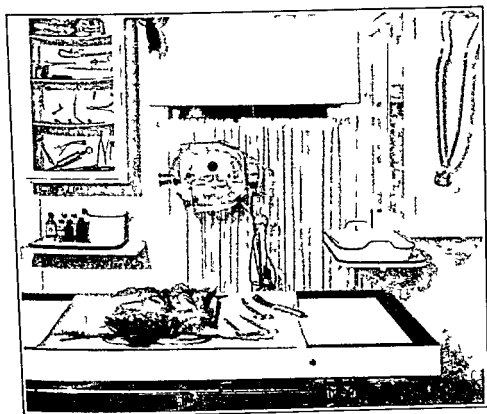


FIG 25 — Many practising veterinarians are well equipped to render expert diagnostic service.

pellicle, as well as odor are noted. If contaminating organisms are present, as indicated by appearance of the colonies, or by examination of stained smears, the growth is transferred to fresh media, and by this diluting process, pure colonies of the organism are often obtained. Certain dyes, added to some media in minute amounts, inhibit the growth

tory in attempting to arrive at a diagnosis. It is felt, however, that some of the more common procedures may be of interest and brief description, of a few of them are given.

The examination given a fowl at the laboratory is essentially the same as that just outlined. Autopsy findings correlated with the history, are in many cases sufficient for making a diagnosis, but unless the changes encountered are definitely diagnostic, further examination is necessary.

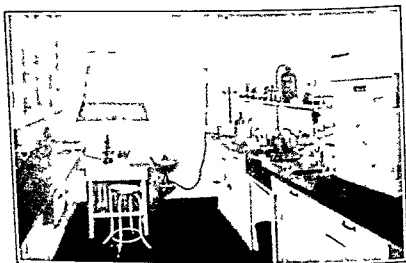


FIG. 24 — A corner of a diagnostic laboratory, showing some of the equipment commonly used

Culturing.—In this procedure, which must be carried out with strict asepsis to prevent contamination, an attempt is made to transfer some of the microorganisms present in an organ, to artificial media (food material) upon which or in which the germs will grow. If the transfer is successful and growth occurs, it enables the detailed study necessary for identification to be made. The "culturing" is effected by transferring some of the material (blood, bit of organ) to the medium by means of a sterile wire loop or sterile forceps.

The cultures are then placed in an incubator in which the temperature is maintained at about 37.5°C . Very few pathogens grow satisfactorily, some not at all, unless the temper-

stained, be very suggestive or even diagnostic of a certain disease condition

Animal Inoculation —Occasionally it is found necessary to determine the effect of suspected organisms or other material when fed to or injected into test animals and for this purpose chickens, guinea-pigs, rabbits and pigeons are commonly used

Examination of Intestinal Scrapings —Examinations of scrapings of such organs as the crop, intestines and ceca are carried out as a matter of routine, principally to detect the presence of small parasites and coccidia. The scrapings are smeared in a film on a glass slide and examined under the microscope, usually without staining

Flotation —This is the method used to concentrate small objects such as worm eggs, coccidia and mites, when they are present in suspected material in small numbers. It is done by placing some of the suspected material in a dense solution of sugar or salt and spinning it in the centrifuge. The high specific gravity of the solution causes the eggs, coccidia, etc., to rise to the top of the fluid, from which the film may be taken and examined. It is particularly useful in the examination of fecal material for the presence of worm eggs

Agglutination Test —Of the biological tests resorted to in the diagnosis of avian diseases, the agglutination reaction is probably the most widely used. It is a highly technical procedure which is extensively used in the diagnosis of pullorum disease. A brief description of the standard (tube) agglutination method may enable the *theory and application* of the test to be better understood

As previously mentioned, certain infections are accompanied or followed by the formation, in the body of the affected individual, of "immune bodies" (antibodies), one type of which is called "agglutinins". These agglutinins are present in greatest concentration in the blood serum and their presence is demonstrable only by the agglutination test. With but few exceptions they are specific for the organism responsible for their production. When serum containing agglutinins is brought in contact with a standardized suspension of the specific microorganism the agglutinins manifest them-

of contaminating germs but do not prevent the growth of other organisms

The behavior of organisms when grown in certain "sugars" is of great help in identification. The ability of the various germs to produce acid or gas or both, in sugars is fairly constant and thus affords a further means of identification. The changes produced in various other substances are also of value in correctly classifying the organism being studied.

If the pathologist suspects the presence of a virus in the specimen the procedure differs somewhat from that followed for isolating and identifying bacteria. Inoculation of susceptible subjects with exudate, suspension of an organ, or their filtrates is a method commonly used and injection of developing chick embryos with bacteria free filtrate is also sometimes used to determine the presence or absence of virus. Certain viruses grow readily in this medium and this enables further study to be made of them. The use of the ultramicroscope, previously mentioned, is certain to become an increasingly useful agent in the field of virus study.

Staining—Many kinds of stains and staining processes may be used in identifying organisms. Their purpose is to bring out in detail the form and stain reactions of the germs and thus to serve as an aid in their identification. The morphology, source, growth, reaction in different media, and staining characteristics of an organism usually enable the bacteriologist to make a positive identification.

The causative agent of tuberculosis (*Mycobacterium avium*) can be grown only by the employment of special technique and media and from two to six weeks are required for the growth to become evident. Because of the fatty or waxy nature of the organism it cannot be stained by ordinary methods. Penetration of the dye into the organism is effected by prolonged staining or by the application of a moderate amount of heat during the staining process. Once the organism takes the dye, however, the color cannot be easily removed even by the application of dilute acids or acid alcohol, hence it is often referred to as being "acid fast."

Direct smears of such material as blood, exudates and suspected tuberculous material may, by being properly

the blood stream. It may be stated, however, that a high titer of the blood serum does not necessarily indicate a severe infection, and that a negative reaction is not an absolute guarantee of the absence of infection. When properly conducted, however, and interpreted by a trained person, the agglutination reaction is one of the most reliable diagnostic tests available.

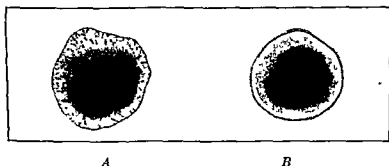


FIG. 26 —The whole-blood, stained-antigen test for pullorum disease.
A, Positive, B, Negative reaction.

Several modifications of the tube agglutination test have been made, and while they are not all strictly laboratory procedures two of the important ones are briefly discussed in this section. The principal advantages of these modifications have been ease of conduct and rapid reactions.

Whole-blood Stained-antigen Test.—This test is being widely used in the diagnosis of pullorum disease and possesses certain advantages which make it the most desirable of the field tests yet devised. The test may be performed in the field, the reactions are rapid, and only one handling of the birds is required. Blood is obtained by snipping the comb at the base of the beak or by drawing a small amount from the large vein on the under side of the wing with a clean syringe and a single drop is placed upon a clean microscope slide. To this drop of blood an equal amount of stained antigen is quickly added and the two mixed by using a clean toothpick. The antigen for this test is a very dense suspension of *Salmonella pullorum* organisms, deeply stained by crystal violet. Positive reactions are usually manifested within one-half to

selves by causing the suspended germs to gather in clumps i.e. to agglutinate. Depending upon the 'dilution' desired the test is performed by placing a measured amount (usually 1 or 2 cc) of the bacterial suspension (antigen) in each of the number of tubes used and adding to these tubes varying amounts of the serum to be tested. The 'dilution' is simply the amount of serum as compared with the amount of antigen in the mixture. If for example there is 1 part of serum and 25 parts of antigen the dilution is 1 to 25; if 1 part of serum is added to 50 parts of antigen the dilution is 1 to 50 and so on.

After the antigen-serum mixtures have been made in the desired dilutions the tubes are incubated for twenty-four to forty-eight hours at 37.5°C and the 'readings' made. A positive reaction is indicated if the bacteria in the suspension clump together and fall to the bottom of the tube with a resultant clearing of the fluid. A negative reaction is one in which no clumping has occurred with the fluid remaining cloudy as it was originally. A partial reaction is one in which only a partial clumping of the organisms and clearing of the fluid has occurred. The titer of the serum refers to the smallest amount of serum which has caused complete agglutination in the dilutions used. In some cases serum titers are very high and this end point can be detected only if the dilutions have been high enough to detect the point at which agglutination ceases.

The reading and proper interpretation of the results of an agglutination test require experience and skill and should be attempted only by those trained to do such work. A positive reaction according to the accepted standards for its determination indicates that the fowl is or has been infected with the organism in question. A negative reaction indicates the absence of such infection. A partial reaction must be interpreted according to the dilution in which it takes place. If it occurs in a high dilution (1 to 500 or 1 to 1000) and there is complete agglutination in the lower dilutions its only significance is an indication of the titer of the serum. If on the other hand it has occurred in a low dilution (1 to 25 or 1 to 50) its significance is difficult of interpretation. It may indicate a beginning infection or it may point to an abatement of the infection and disappearance of agglutinins from

Hemagglutination (HA) Test.—This test (IIA) is employed for the detection and identification of certain viruses such as Newcastle disease, fowl plague and bacteria such as pleuropneumonia-like organisms (PPLO). In the case of a virus developing chicken embryos are injected with suspected material and after a few days allantoic fluid is collected and, in varying dilutions, mixed in tubes with avian red blood cells



FIG. 28 —Diagnosis of virus infections may require inoculation of tissues into embryonated chicken eggs (Courtesy of Minnesota Agricultural Experiment Station)

The mixture is allowed to stand at room temperature for about thirty minutes. The manner in which the red cells settle on the bottom of the tube determines whether the test is positive or negative. In the identification of PPLO the bacteria are grown in broth cultures and the bacterial growth is used in place of allantoic fluid in conducting the hemagglutination test. The HA test is considered very reliable in differentiating Newcastle disease virus from those of infectious bronchitis and laryngotracheitis.

Hemagglutination-inhibition Test (HI)—This procedure (III) is used to determine the presence of hemagglutination-inhibiting antibodies in the serum of birds that have recovered

one minute by a clumping or agglutination of the stained organisms. If the test is negative, the mixture of blood and antigen remains homogeneous and no clumping occurs.

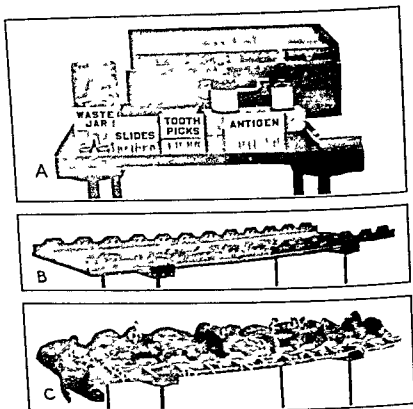


FIG. 27.—Field equipment for making the whole-blood stained-antigen test for pullorum disease. The specially-constructed table shown here is used to hold the fowls until the results of the test are known. (After Barger and Torrey 1933.)

The Rapid Plate Agglutination Test.—This test is similar to the whole-blood method, except that serum is used instead of whole blood, and the test is made on a glass plate ruled into small squares. The antigen for this method is also very dense but usually contains no dye. The amounts of serum and antigen are carefully measured by pipetting, enabling any desired dilutions to be made.

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from Newcastle disease fowl plague or chronic respiratory disease To definite amounts of standardized virus or bacterial suspension are added varying amounts of suspected serum the mixture is allowed to stand in tubes for a few minutes and a specified amount of red blood cells are added If the serum of the suspected bird contains no antibodies for the virus (NDV) or bacteria (PPLO), the red blood cells settle to the bottom of the tubes in a given manner but if antibodies are present the settlement of the red cells follows a different pattern

Serum Neutralization Test (SN) — This test (SN) is a standard procedure that is widely used in identifying viruses Serum from an immune bird contains antibodies capable of neutralizing a virus and the test is made by mixing definite amounts of suspected serum and known virus, then injecting the mixture into developing chicken embryos or susceptible chicks or poults Failure of the mixture to cause disease in either the chicks or embryos would indicate that the suspected serum came from a recovered or immune bird This test (SN) is used to identify a number of virus diseases such as Newcastle disease, infectious bronchitis, laryngotracheitis, fowl pox and fowl plague

Isolation of Virus — For this test, suspected tissue is collected from the bird and prepared for injection into developing chicken embryos of nine to ten days If virus is present, it will grow in the fluids and in the case of some viruses the embryos will die within five or six days and lesions may be noted The fluids from dead or living embryos are subjected to various tests to identify the virus

The foregoing outline of laboratory procedures represents only a few of the methods used in making a diagnosis. In some instances detailed technique is necessary before drawing final conclusions, but it is hoped that the examples given may aid the reader in interpreting the reports of findings and reactions as they may come to him from the laboratory

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differences among breeds. Extensive tests conducted in flocks in New Jersey, Connecticut, Massachusetts, and North Carolina where only natural selection had been operating, showed far fewer reactors in White Leghorns than in the heavier breeds. In controlled experiments in which chicks of several strains of Leghorns and of heavy breeds were used it was noted that the former were always more resistant to artificial inoculation as well as to natural exposure to *S*

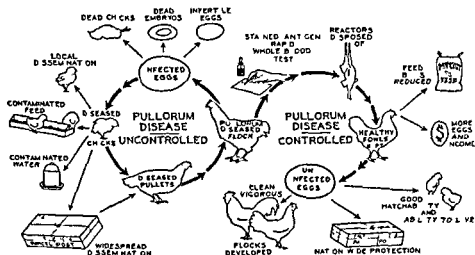


FIG. 29.—Diagrammatic comparison of serious effects of uncontrolled pullorum disease with the benefits resulting from the use of the agglutination test and the disposal of reactors (Courtesy of U S Department of Agriculture)

pullorum. These authors conclude that resistance to pullorum disease is a characteristic of the Leghorn breed and that it is not necessarily associated with the dominant white which differentiates the white variety from others.

Cause—The disease is caused by the microorganism *Salmonella pullorum*, which was first recognized as the causative agent by Rettger in 1900. *S. pullorum* is found in practically all of the internal organs of affected chicks and poults, particularly the liver, lungs, yolk sac, intestinal tract, as well as in the blood. In mature hens and turkeys the ovary is the common site of localization although occasionally the organism is found in the lungs, liver and intestinal tract. Many of the eggs laid by diseased birds carry

Chapter 6

BACTERIAL DISEASES

MANY of the more common and economically important diseases of fowls are caused by bacterial agents. Fortunately these infections, as a rule, are not transmissible to other farm animals although there is a notable exception in the case with which swine may contract avian tuberculosis. The important diseases in this group include pullorum disease, tuberculosis, fowl cholera and fowl typhoid. There are several others of less importance. Because of the prevalence and importance of pullorum disease it is discussed separately from the other *Salmonella* infections.

PULLORUM DISEASE

Pullorum disease is primarily a disease of baby chicks, although it is often found in mature hens as a chronic ovarian infection and occasionally in the pericardial sac and testes of mature males. Poults and mature turkeys are susceptible, with the manifestations in this species being practically the same as in chickens. The disease has also been found occurring naturally in pheasants, ducks, European bullfinches, sparrows guinea fowl, baby quail, canaries and pigeons. Among mammals, rabbits appear to be readily susceptible and the infection has been reported as occurring in hogs, foxes, mink, and man.

In baby chicks and poults, the disease assumes an acute, septicemic, highly fatal form, and most of the losses occur during the first two or three weeks of life. In adult fowls the infection is usually symptomless although it may occasionally be manifested by diarrhea, depression, lack of appetite, and paleness of the comb and wattles.

All breeds of chickens appear to be susceptible although variations in the degree of susceptibility are found among the various breeds. In studies of breed susceptibility to infection with *S. pullorum*, Hutt and Scholes (1941) found significant

Other conditions affecting baby chicks and poults may induce symptoms which are indistinguishable from those mentioned.

In adult fowls no symptoms, as a rule, are observed. Mature hens and turkeys shown by autopsy and bacteriological findings to be carrying *S pullorum* infection in the ovary usually give no indication whatever that they are diseased. Occasionally, however, manifestations of an acute or chronic form of the disease are seen in mature birds. In acute cases, in which the infection has been recently acquired or is a flare-up of an already existing condition, there is weakness, the feathers are ruffled, and a greenish brown diarrhea is present. There may also be paleness of combs, listlessness, depression, and droopy heads and wings.

Postmortem Appearance—In chicks and poults which die soon after hatching, no significant changes may be present. Those which survive for a longer time, however, usually show definite, and in some cases marked, alterations of the organs. The crop is empty, and the liver shows a peculiar, streaky, brick-red discoloration in contrast to the normal yellow appearance during the first few days of life. Inflammation and thickening of the heart sac and the presence of small, grayish nodules in the wall of the heart are seen in many cases which survive for any length of time. Pneumonia is not uncommon in birds dying early, with firm grayish nodules appearing in the lungs within a few days. In some cases the affected birds appear to be bloated, the swelling being caused by the accumulation of a gelatinous substance under the skin and in the body cavities. The presence of an unabsorbed yolk sac is not diagnostic of pullorum disease. During the first few days of life the yolk-sac serves as a source of food material and is found in all chicks and poults. If, however, it persists after three or four days in an unabsorbed, shrunken, discolored state, pullorum disease may well be suspected. In some cases the principal locus of infection appears to be the intestines which show small, necrotic foci, particularly in the large portion of the bowel.

In mature fowls the organic changes are confined principally to the ovary. Instead of the ova appearing round, plump, and golden yellow as they normally do, those which are visibly diseased are shrunken, misshapen, have a greenish

An unusually high mortality in baby chicks or poults during the first two weeks after hatching immediately suggests pullorum disease as the cause, and lesions observed in affected individuals are often strongly suggestive of pullorum infection. Since similar symptoms, lesions and high mortality may result from other infections and even from faulty management practices, the only criterion of infection with *S. pullorum* is the isolation and identification of the organism. Even the agglutination test has its limitations in that fowls infected with fowl typhoid will react to this test, as may some that are infected with paratyphoid organisms.

The agglutination test used for the detection of adult carriers of pullorum infection, may be applied in three ways—the tube agglutination test, the rapid whole-blood plate test, and the rapid serum plate test.

The test makes use of a liquid suspension of killed pullorum organisms known as an antigen. The presence of *S. pullorum* in the blood of an infected fowl causes the formation of immune substances called antibodies. When the antigen is mixed with either the whole blood or the serum of an infected fowl, these antibodies cause the killed organisms in the antigen to clump together in small masses which are easily seen. No such clumps form in the blood of noninfected fowls because there are no antibodies present.

The rapid tests are much more widely used than the tube test because they are adapted to field conditions and permit the immediate identification of carrier fowls.

A variant type of pullorum found in Canada in 1941 and later in many parts of the United States has made it necessary to use a polyvalent type of antigen where the variant type of the disease is known to occur or is suspected.

Dissemination—The enormous traffic in baby chicks has undoubtedly played a major rôle in the widespread distribution of pullorum disease. It has been demonstrated that the infection is readily spread in forced-draft incubators at hatching time. Chicks hatched from infected eggs may harbor the bacteria in the down which, upon drying, becomes loose and fluffy, being readily blown to various parts of the incubator even by light drafts of air. Susceptible chicks or poults breathe in, or possibly ingest, these small contaminated

or leaden color, and the content varies from a soft consistency to one of firmness as if the yolk material had been cooked. There may be enlargement of the heart and small, grayish, firm nodules in its wall.



FIG. 30 — A chick showing characteristic lung lesions of pullorum disease (Courtesy of Purdue Agricultural Experiment Station.)

Diagnosis — There are several aids to the correct diagnosis of pullorum disease but positive proof of the presence of the malady depends upon the isolation and identification of *S. pullorum*.

the dry chicks should be removed. It is suggested that only two fumigations are necessary with this method, the first one being carried out when one-tenth to one-fifth of the chicks are out of the eggs, and the second, twelve to fifteen hours later.

The importance of maintaining relatively high humidity at hatching time has been stressed by many investigators. Wet-bulb readings of 90° to 95° F. decidedly reduce the circulation of chick down as compared with the amount of circulating particles when the wet-bulb reading is lower.

Regardless of the method employed, any attempt to disinfect incubators by fumigation, regardless of the degree of efficiency attained, is at best but a means of reducing the amount of spread in those cases in which eggs from infected and non-infected fowls are hatched in the same machine. *The source of infection still remains in diseased, mature fowls.*

The rate of spread of pullorum infection among mature birds varies greatly. In the past, some have considered the male bird as an important factor in transmitting the infection from diseased to non-infected females, but experimental work done on this phase of the problem indicates that the male is not a factor in this connection. Attempts to communicate the disease by placing reacting males in the same pen with non-reacting females have not been successful.

It has been shown that in some cases, infected birds eliminate *S. pullorum* in their feces and this mode of dissemination must be taken into consideration in dealing with the disease. Carefully conducted tests indicate that the infection may be spread among mature fowls by contact and cohabitation and it appears that the more closely fowls are confined the greater is the opportunity for such dissemination.

Mortality.—There is definite evidence that pullorum infection increases embryo mortality and thus decreases the hatchability of eggs. Studies have shown that the percentage of dead embryos is greater in eggs from reacting fowls than from non-reactors.

In baby chicks and poults the heaviest losses occur during the first two or three weeks of life with the mortality ranging from a small percentage in a few cases to as high as 100 per

finding that 90 per cent of the surviving chicks reacted positively to the whole-blood test at twelve weeks of age. Anderson *et. al.* (1948) concluded that sodium sulfamerazine administered in the drinking water at a concentration of 0.2 per cent for a period of seven days afforded considerable protection to artificially inoculated chicks.

In trials with three lots of yearling hens, Cole (1948) fed an all-mash ration containing sulfamerazine 0.5 per cent, sulfadiazine 1.0 per cent, and sulfaguanidine 1.0 per cent respectively, during alternate, approximately weekly periods for four months. The results indicated that the prolonged feeding of these drugs did not prove effective in removing *S. pullorum* from the birds. Survivors were found to give positive reactions to the agglutination test at the end of the trials and for 185 days subsequent to the termination of feeding the drugs.

Chang and Stafseth (1950) and Cooper and co-workers (1951) reported similar results. The use of drugs must therefore be considered as of only secondary importance in dealing with pullorum disease.

It is possible, of course, that some new drug may be found which will be effective against bacteria of the *Salmonella* group. Thus Smith (1954) and Wilson (1955) found furazolidone to be partially effective in eliminating *S. pullorum* from chicks which had been artificially infected at one day of age. Well-established infections are much less susceptible to treatment.

Control.— *Any plan for controlling pullorum disease must have as its principal objective the elimination of the most prolific and constant source of infection—the carrier hen or turkey.* One phase of the National Poultry Improvement Plan is concerned with the control of pullorum disease. Practically all states have control programs which are being carried on in cooperation with the National Plan. Local conditions may make it necessary to alter some of the details of any pullorum disease program but available records indicate that good progress is being made particularly in certain sections of the country.

It has been demonstrated that pullorum disease can be

cent of the lot. Losses ranging from 5 to 15 per cent have been reported by Hinshaw (1948) in poults at 9 to 10 weeks of age which had survived earlier outbreaks.

Effect on Production.—Studies made of the records of reacting and non reacting fowls show clearly that pullorum disease causes a definite lowering of production. The records of Asmundson and Biel (1930) for 689 hens (587 negative and 102 positive reactors) of six breeds in the first year of production showed that the average for the disease-free group was 221 eggs as compared with 160 eggs for the infected lot. Kernkamp (1932) recorded the production of 209 fowls (123 positives & 86 negatives) selected without reference to type breed variety fecundity or age with the majority of them being in the second laying season and found that the average production of the non reactors was 87 per cent higher than that of the reacting fowls.

Treatment.—For many years efforts have been made to find an effective medicinal treatment for pullorum infection and many drugs have been tried in this connection but they have almost without exception been ineffective regardless of the substance used or its method of administration. With the advent of the sulfonamides however it appears that some reduction in the mortality rate in infected birds may be effected by the administration of certain drugs of this group.

Seven sulfonamides were tested by Severens Roberts and Card (1945) with respect to their ability to reduce mortality from pullorum disease. Sulfadiazine and sulfamerazine were found to be most effective as judged by both mortality and rate of gain of surviving chicks. In a natural outbreak of pullorum infection in baby chicks Anderson (1946) reported that 0.5 per cent sulfamerazine in the dry mash over a period of 21 days was definitely beneficial and states his belief that the drug is effective in controlling natural outbreaks of the disease if an early diagnosis is established and treatment promptly instituted.

In additional tests upon baby chicks Bottorff and Kiser (1947) found sulfadiazine sulfamethazine and sulfamerazine to be equally effective in reducing mortality as much as 30 to 56 per cent with no significant difference in the weight gains after twenty-one days. They also made the interesting

produce disease in other animals. The tubercle bacillus was discovered by the German scientist Koch, in 1882—a discovery destined to be of tremendous importance to mankind. Since that time, studies have shown that there are three principal types or strains of the organism; *viz.*, human, bovine and avian, distinguishable by their ability to attack the different species of animals and by laboratory procedures.

The avian tubercle bacillus is resistant to external influences and under certain conditions may survive outside the body of an infected fowl for a long time. Schalk (1928) found that rigorous winter weather did not destroy the organism, and that carcasses of tuberculous birds buried in manure and in varying depths of soil contained virulent organisms after three, six, nine, and in one case after twelve months of such exposure. Once infected, a poultry yard may remain a source of the disease for a long period, even after the removal of tuberculous fowls. The tubercle bacillus is readily destroyed by direct sunlight, but the latter is effective only on the surface of material containing the germs, and not when the organism is buried in soil, manure or débris. Chemical disinfectants are effective against the tubercle bacillus when it is present in material which contains no protein. Moist heat destroys the organism rather rapidly, but as mentioned, cold does not readily affect it.

Occurrence.—Tuberculosis in fowls in the United States was apparently first reported by Bray in 1896; and Pernot in 1900 was the first to confirm its presence by bacteriological examination. The disease is at present prevalent in this country, especially in the North Central portion.

Susceptibility.—Several species of fowl are susceptible to avian tuberculosis, but the disease is far more prevalent in chickens than in other birds. This probably is not because of any increased susceptibility of chickens, but because of the closer contact and greater opportunity for spread among this species. The infection has been observed also in turkeys, ducks, geese, pigeons, parrots, canaries, pheasants, sparrows, swans, guinea-fowls, cow-birds, cranes and crows. The disease is apparently not widespread in turkeys and when these birds are affected it is usually through cohabitation with infected chickens. It has also been shown that the

successfully combated by rigid adherence to the following measures

1 *Systematic testing of all fowls in the flock* The frequency with which the flock should be tested must be determined by the degree of infection encountered. If no reactors are found it is obvious that every effort should be made to prevent introduction of infection into the flock. When reactors are found it is important that they be removed at once. It is equally important that frequent tests—monthly if possible—be conducted thereafter until all remaining birds give two successive negative tests. To allow as much as six months to elapse after testing the flock and removing reactors before making another test, is to invite spread of the disease by birds which have become infected in the meantime. After a pullorum-disease-free status has been established for a flock, future tests should be conducted at intervals of no more than six months.

2 *Procure eggs only from sources known to be disease-free* The fact that pullorum disease is transmitted through the egg renders the necessity for procuring eggs from a clean source so obvious as to require no argument.

3 *Cleanliness at all times about the premises* is important and it is especially basic that a thorough cleaning and disinfection be carried out following removal of reactors. Unless this is done there is the possibility of disease-free fowls becoming infected by organisms which may be present in the litter, on the floor, or on various pieces of equipment. Special attention should be given to incubators, brooders, and all equipment to be used for baby chicks.

FOWL TUBERCULOSIS

Tuberculosis of fowls is a chronic, infectious disease, very similar to tuberculosis of other animals. It is of common occurrence, and in some localities it causes enormous losses to flock owners.

Cause—Avian tuberculosis is caused by the acid fast microorganism *Mycobacterium avium*. This germ is closely related to the tubercle bacillus of man and of animals. While it is most commonly found in fowls, it is also able to

the head become pale, and the feathers are ruffled. There is evidence of increasing weakness and the affected fowl moves about very little, being listless and unthrifty. The appetite is sometimes retained until a short time before death. Lameness in one or both legs is commonly observed, and is caused by the development of tuberculous abscesses in the joints.

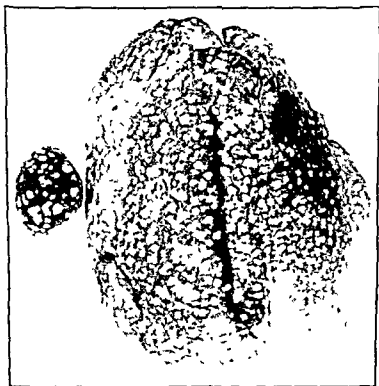


FIG. 32.—A tuberculous liver and spleen. Nearly all fowls suffering from tuberculosis show lesions in the liver. (Courtesy of Illinois Agricultural Experiment Station.)

The wings may be similarly affected, and when involved hang in a droopy position. Diarrhea often appears in the later stages, followed by complete exhaustion and death. Well-advanced cases of the disease are occasionally found in fowls in good condition, but in the great majority of cases, emaciation is a constant finding.

Postmortem Appearance.—The characteristic lesions of tuberculosis in fowls, as in other animals, are tubercles.

intestinal canal, the feces of such a fowl being a constant source of infection to the remainder of the flock. As a rule, avian tubercles are not gritty when cut, although some of the older ones have a granular consistency which can be detected upon drawing a knife through them. The cut surface of a typical tubercle usually appears gravish and shining, but in older lesions may appear yellowish in color and cheesy in consistency.

Tubercles in the liver and spleen are easily removed in their entirety from the surrounding tissue, but those involving the intestine and mesentery are not easily enucleated.

Diagnosis — Emaciation is a rather constant symptom of tuberculosis, but because it also is a symptom in other diseases it is not sufficient evidence upon which to diagnose the condition. The presence of characteristic tubercles in the liver, spleen and intestine is very suggestive, but care should be taken not to confuse these lesions with those occurring in lymphomatosis and blackhead. In the latter disease the involved areas in the liver appear sunken and ulcer-like, while in avian tuberculosis the lesions are raised above the surface of the organ, giving a lumpy or beaded feeling upon palpation. The nodules in the intestinal wall caused by one variety of tapeworm might be mistaken for those of tuberculosis, but the absence of characteristic lesions in the liver and spleen, and the presence of tapeworms in the bowel should enable a distinction to be made. Swollen joints should always be regarded with suspicion, especially in pigeons.

Laboratory diagnosis is made by microscopic examination of affected tissue, by injection of suspected material into susceptible animals, and in some cases by growing the organism by special technique upon suitable media.

Tuberculosis in living fowls may be diagnosed by the use of tuberculin. This substance is made by growing avian tubercle bacilli in fluid media for several weeks, after which they are killed by heat and removed by filtration. This leaves a sterile clear, brownish fluid containing the growth products of the organism, which after standardization constitutes tuberculin. It cannot cause the disease, but when properly injected into tuberculous fowls induces a reaction which is diagnostic. The test is made by injecting,

with a small hypodermic syringe a minute amount (1 small drop) into the skin of one wattle, the other being left as a control. The proper injection of tuberculin requires skill and care. The point of the needle must not be pushed through the skin because tuberculin deposited under the skin does not give the desired reaction. On the other hand, the injection must not be made too superficially as the skin is likely to rupture under the pressure.



FIG 34—The wattle test for tuberculosis. The swollen wattle is evidence of infection.

The results of the test are best read forty-eight hours following injection. Many reactions will appear within twenty-four hours but some are slower in becoming evident, therefore in order that none be missed by reading too early, forty-eight hours should elapse after injection before final readings are made. A positive reaction consists of a doughy swelling of the injected wattle. They vary greatly in size and appear blanched as compared with the red color of the normal wattle.

The success of the test depends largely upon the skill of the operator and the potency of the tu-

berculin used. Occasionally an advanced case of the disease does not give a reaction, but such birds usually manifest clinical symptoms which are suggestive, and which should be considered in making the diagnosis.

A rapid agglutination test for the detection of tuberculosis in fowls has been reported by Moses, Feldman, and Mann (1943) and by Karlson Zinober and Feldman (1950). Richev,

Mack and Stafseth (1954) have summarized their own and earlier work on the hemagglutination reaction in avian tuberculosis

Dissemination — The greatest single factor in the spread of avian tuberculosis is the fecal material of infected birds. As previously mentioned, open tubercles or ulcers in the intestine are continuously discharging virulent organisms into the bowel and these are passed to the outside in the droppings. Fowls constantly pick up particles from the ground and are thus exposed by ingesting food and water contaminated by the feces of tuberculous birds. After being taken into the digestive tract, the organisms pass through the wall of the intestine and are carried to the liver where they lodge and produce well-defined lesions. From the liver they are transported to other parts of the body by means of the blood stream and the infection thus becomes generalized.

The introduction of the disease into a flock may occur in several ways. Affected fowls may be added to the flock, the birds may have access to premises inhabited by tuberculous birds, and diseased wild flying birds may transmit the infection while feeding in the poultry yard. It is also possible for man, as well as other animals, to carry the infection mechanically from one place to another. Feed sacks to which tuberculous fowls have had access when returned to the mill or other distributing points for further use might well be considered a potential means of disseminating tuberculosis as well as other diseases.

It has been established that occasionally an egg laid by an infected hen contains tubercle bacilli, but the incidence is so small that spread of the disease through the egg may be considered as practically negligible. Harshfield, Roderick, and Hawn (1937) examined by tuberculin test or by culturing the livers and spleen, a total of 1141 chicks hatched from reacting hens and in no instance was tuberculosis found. Eggs infected with tubercle bacilli have a lowered hatchability rate, and the possibility of dissemination by this means is thus further reduced.

Schrick (1928) was able to induce tuberculosis in one case by feeding *fl. larvae* which had developed in a tuberculous carcass, to susceptible birds. This probably is not an impor-

should never be thrown out where other fowls or hogs may have access to them

Following disposal of the flock it is best, after a thorough cleaning and disinfection of houses and equipment, to allow the premises to remain idle for several months, during which much of the remaining infection will have died out. Scalding hot water containing 3 per cent of common lye, compound cresol solution in 3 per cent dilution, and 10 per cent formalin are all inexpensive disinfectants and are very effective when applied freely and thoroughly to surfaces from which all dirt has been removed. Ordinary quicklime sprinkled over the yard aids in reducing the infection upon the surface of the soil.

If only a few fowls in the flock give reactions to the tuberculin test, the infected birds should be properly disposed of and the houses and equipment thoroughly cleaned and disinfected. Discing of lots and spading of soil inaccessible to the disc should be carried out wherever possible. A subsequent tuberculin test within six months is also advisable to detect any birds which failed to react initially.

If birds are purchased for restocking, they should come from a source known to be free of infection.

Feldman (1939) and McCarter, Hastings and Beach (1940) have called attention to the rôle of avian tubercle bacilli in sensitizing cattle to mammalian tuberculin. The former author states, "It seems not unlikely that in the United States the avian tubercle bacillus sensitizes more cattle to mammalian tuberculin than is generally recognized, and the possibility is suggested that at least some of the so-called non-visible lesion reactors react to mammalian tuberculin as a consequence of sensitivity induced by avian tubercle bacilli."

Because of the slow development of avian tuberculosis, the disposal of all fowls over two years of age will serve as a means of reducing the incidence of infection in the flock. Unless it is desired to retain the birds as breeders, a consistent selling program will also serve to eliminate the older fowls at a time when their production normally begins to decline. The danger of allowing fowls to run with swine has been emphasized.

Once tuberculosis has occurred on the premises, constant care should be exercised to detect its recurrence. The tuber-

age on commercial duck farms. Among laboratory animals rabbits succumb readily to inoculation. In geese the disease often assumes an extremely virulent character and is commonly referred to as goose septicemia. The domestic animals are, in general, resistant to the fowl type of the organism.

Symptoms—If the disease is very acute, the first indication of infection is the finding of birds dead under the roost or on the nest and in which no symptoms were visible only a short time before. Extremely fat fowls are particularly susceptible to this peracute form of the malady. In the less acute type a greenish or yellowish diarrhea is often the first indication observed. As the disease progresses the birds become listless and sleepy, dislike to move, and the feathers are ruffled. The appetite is impaired but increased thirst is often noted. Respiration is rapid and the accumulation of mucus in the upper air passages causes a rattling noise as the fowl breathes. The body temperature is increased and the comb and wattles become purplish in color. Edema or swelling of the wattles is not uncommon and the feathers about the vent are smeared by the profuse diarrhea. In some of the outbreaks of fowl cholera in young ducklings reported by Hilbert and Witter (1936) more than 60 per cent of the affected birds showed localization in the leg, with resultant abscess formation involving almost all of the unfeathered portion of that area.

The chronic type of the disease usually occurs in birds which have survived an outbreak. Chronically affected fowls gradually become emaciated, appear depressed, and the combs, wattles and membranes of the head are pale. Fowls suffering from this form of the disease may linger for several weeks before succumbing. Lameness occasionally is observed as a symptom in prolonged cases and when present is caused by localization of the organisms in the joints, resulting in an abscess.

Delaplane and Higgins (1948) state that the respiratory form of fowl cholera is the most important respiratory disease of bacterial origin in chickens in some states. It is usually seen in farm flocks rather than on commercial poultry farms because of the common practice of mixing young and old birds together when the pullets reach laying age.

was prolonged, the ova were soft, flabby, irregular in outline and pedunculated. The yolk membrane was very weak, being readily ruptured, and occasionally a greenish colored ovum was observed. The author did not consider these alterations to be characteristic of fowl cholera, however, since similar changes are observed in hens affected with pullorum disease and fowl typhoid.

Diagnosis—The sudden death of birds in an apparently healthy flock is strongly indicative but not diagnostic of fowl cholera. If, in birds which die suddenly, numerous hemorrhages are present on the membranes of the body, particularly on those of the heart, fowl cholera may well be suspected. Greenish diarrhea may also be considered as pointing to the presence of the infection.

Positive diagnosis of fowl cholera is dependent upon finding characteristic organisms in stained smears of heart blood or upon isolation of them from the heart blood, liver or other organs. These procedures are of necessity limited to a laboratory, to which suspected specimens should be submitted in case of doubt. Certain biological tests have been tried in the diagnosis of fowl cholera but in general the results have not been satisfactory. The need for a dependable diagnostic test is definite, particularly so if it could be relied upon to detect carrier birds which act as reservoirs of infection in flocks. Shook and Bunyea (1939) describe a whole-blood stained antigen agglutination test which gave excellent results and which was in very close agreement with tube tests. By applying this test and removing reactors these workers were able to control the infection in a flock in which the disease had been prevalent.

The use of this newly developed antigen has been limited however, and further work is being done toward improving the product. The production of the antigen is covered by a United States patent issued to the Secretary of Agriculture from whom permission would be required before it could be produced commercially.

Dissemination—*P. multocida* is widespread in Nature and may be present in the respiratory tracts of birds without inducing symptoms. When, however, the resistance of fowls harboring the organism is lowered by any means such as

of 0.1 per cent or even 0.05 per cent in the mash might help in preventing enzootic *Pasteurella* infections of the respiratory tract in chickens providing further studies proved the drug non-toxic at the levels used. Kiser *et al* (1948) were able to significantly reduce mortality in both experimentally infected chicks and in those naturally affected, by as much as 83 per cent in the former and 75 per cent in the latter. The drug was tried by incorporating 0.5 to 1.0 per cent in the feed and 0.1 per cent in the drinking water. Queen and Quortrup (1946) found penicillin effective in protecting wild ducks experimentally infected with pasteurellosis.

McNeil and Hinshaw (1948) found that dosages of 150,000 units of streptomycin prevented mortality from *P. multocida* in turkeys if treatment was given before or at the time of inoculation. When treatment was delayed six to twenty-four hours, there were carriers and also a large number of cases of joint involvement.

Vaccination—Despite the fact that Pasteur was able to induce immunity to cholera in fowls by the use of vaccines containing the causative organism, subsequent attempts have not been so successful. Pasteur's results may have been attributable to the use of a strain of organisms possessing unusual powers of immunization, as great variation in this respect has been noted by investigators. Opinion is not unanimous regarding the use of vaccines for the prevention of fowl cholera but, in general, their employment has not been considered to be of dependable value in controlling the disease. Hilbert and Tax (1938), however, were able to obtain excellent results by the use of a vaccine in reducing losses from cholera among ducks. Gibbs (1940) also reported favorable results from the employment of a vaccine in reducing the number of deaths among ducks on a farm on which the disease was taking a heavy toll. In studies of the disease in wild ducks, Queen and Quortrup (1946) were able to protect wild ducks against experimental fowl cholera infection by use of an autogenous bacterin. These reports are encouraging and if further work establishes that vaccines have a constantly dependable action in preventing fowl cholera, a great service will have been rendered the industry.

Antiserum capable of protecting fowls for a short time has

organism is not resistant and is readily destroyed by the common disinfectants. Direct sunlight is quickly destructive to *Salmonella gallinarum*, but when buried in the soil, the organism may survive for some time.

Occurrence—Fowl typhoid is widely disseminated and apparently occurs wherever poultry is kept to any extent. There is evidence, however, that the disease is less prevalent in some sections than formerly, but no adequate explanation has been offered for the apparent decline in these areas. Field observations indicate that warm, wet weather is conducive to occurrence of the infection.

Susceptibility—Chickens and turkeys appear to be the most susceptible of all fowls, but the disease has been reported in pigeons, pheasants, ducks, quail, grouse, guinea fowls and sparrows. Hinshaw (1930) reported that fowl typhoid was apparently the cause of greater losses in California turkeys than was blackhead. Johnson and Anderson (1933) reported an outbreak in a flock of 100 guinea fowls in which the mortality was about 30 per cent. Whereas formerly the infection was believed confined to mature birds, it has been shown to attack young chicks and in some cases to result in considerable loss.

Symptoms—Droopiness is often the first symptom of fowl typhoid infection. The affected bird is listless, the head is drawn in and the wings are allowed to sag. The comb and wattles are usually pale, the feathers are ruffled, and there is a profuse greenish diarrhea. The course of the infection is from two to ten days in acute cases, while birds chronically affected may live for several weeks and show few, if any, visible symptoms.

Postmortem Appearance—Paleness of the visible membranes is a rather constant finding in fowls dead of fowl typhoid. The liver is enlarged, dark in color and often possesses a greenish sheen. Tiny grayish spots may be present on the surface of the liver. The spleen is often enlarged, as are the kidneys. Marked changes are frequently noted in the heart and consist of grayish, firm nodules of varying size in the heart wall. The ovary may be involved especially in chronic cases, and may have the same general appearance as in pullorum infection. A low-grade inflammation of the

been produced but principally because of the cost of the product and the short duration of protection it has not been used to any extent.

Prevention and Control The presence of fowl cholera in a flock calls for vigorous measures of control. Dead and ailing birds should be removed immediately and destroyed by burning. All utensils should be disinfected daily and the houses frequently. If it has not already been done the feed and water troughs should be placed so as to prevent their contamination by infected droppings. Because of the rapidity with which the disease spreads it is not advisable to attempt isolation except in large establishments where every effort should be made to confine the outbreak to the unit in which it appears. The concentrates in the ration especially the protein portion should be reduced at least one half even at the expense of lowered production. Birds to be added to the flock should be quarantined for at least two weeks to preclude the introduction of fowls in the incubative stage of the disease and the presence of wild flying birds should be reduced to a minimum. The sale of apparently healthy birds from an infected flock is not to be encouraged because of the obvious danger of disseminating the disease to other groups of fowls and thus creating new centers of infection.

FOWL TYPHOID

Avian typhoid is an infectious septicemic disease of poultry readily communicable and usually acute in character although chronic cases are not uncommon. The disease was first described in this country by Moore in 1897 although it had previously been reported in England by Klein in 1889. Moore designated the condition infectious leukemia but later studies indicated that the leukemic condition of the blood though characteristic of fowl typhoid was not a disease entity.

Cause—The causative agent of the disease is the microorganism *Salmonella gallinarum*. It is so closely related to *Salmonella pullorum* that the two are interagglutinable; i.e., serum which agglutinates *Salmonella pullorum* antigen will also agglutinate *Salmonella gallinarum* and *vice versa*. The

exists, by wild flying birds and probably by other unwelcome visitors to the chicken yard

Supportive evidence that the infection can be transmitted through the egg has been obtained by many workers. Beaudette (1925) isolated the organism from the heart blood of baby chicks and from unabsorbed yolk-sacs in five-week old chicks and interpreted these findings as pointing to transmission of the infection from hen to chick through the egg. Beach and Davis (1927) concluded that *S. gallinarum* may produce an acute, highly fatal disease of young chicks and transmission through the egg was suggested by their studies. These authors also state that *S. gallinarum* produces ovarian infection in hens indistinguishable from that caused by *S. pullorum* except by cultural studies. Hinshaw and Taylor (1933) reported a case of ovaritis in a turkey hen from which *S. gallinarum* was isolated and state that their findings also point to transmission through the egg. Boney (1947) suggests transmission of typhoid infection in turkeys through the egg as a result of his experiments and found the organism to be readily isolated from the reproductive systems of both males and females.

Mortality—The death loss in different outbreaks of fowl typhoid varies greatly. The disease may assume a rather mild character in which few birds die and many recover, or it may be so severe as to cause a high death-rate.

Treatment—Some of the sulfonamides have reduced mortality when given continuously or intermittently in the feed or drinking water, but losses often recur when treatment is stopped. More recently Grumbles, Wells and Boney (1954) and Lucas (1955) have reported excellent results from the use of furazolidone in low concentration—100 grams to a ton of mash.

In the case of small flocks the disease may be eradicated by depopulation, followed by thorough and complete disinfection of the premises before restocking.

CHRONIC RESPIRATORY DISEASE COMPLEX

This disease has been reported in chickens and turkeys (see Infectious Sinusitis) from many sections of the United

intestinal lining is usually observed and the intestinal contents are slimy and yellowish. The blood is thin and watery and clots slowly or not at all.

In young guineas dead of fowl typhoid, Beaudette (1938) noted dusky lungs, acute swelling and mottling of the liver, acute swelling of the spleen, catarrhal inflammation of the intestines, and mucus in the proventriculus, nasal cleft and upper trachea.

Johnson and Pollard (1940) found in poultz which had died of fowl typhoid large, liquid, retained yolks, slightly enlarged, friable livers which were light in color and the surfaces of which were mottled with hemorrhagic areas. There was slight congestion in the duodenum and crop, and practically no food in the alimentary canal.

Diagnosis — As with many other bacterial diseases, definite diagnosis of fowl typhoid can be made only by laboratory procedures. The symptoms and lesions enumerated are highly suggestive, however, and in the absence of laboratory assistance must be depended upon to identify the condition. The greenish hue of the liver is a rather characteristic finding, and the paleness of the comb, wattles and membranes is a helpful guide. The hemorrhagic spots and marked inflammation of the intestines seen in fowl cholera are absent in fowl typhoid. The less acute course of the disease also helps to differentiate it from fowl cholera, as well as from some of the more chronic conditions such as tuberculosis and parasitic infestation.

Diagnosis in the field might be aided by the use of the rapid agglutination test for pullorum disease, since both conditions give positive reactions to the test, but possible confusion in attempting to differentiate the two would appear to minimize the usefulness of the test in this connection.

Dissemination — The droppings of infected fowls teem with the causative organism and it is through contamination of the soil, water and feed by such birds that spread takes place. Recovered, carrier birds constitute a constant source of infection. Introduction into the flock may be brought about by newly acquired fowls, by contaminated crates and sacks by humans who have come from premises where the infection

source of infection for natural outbreaks of the disease. The disease must be considered air-borne and contamination of feed, water and litter may be additional sources of the infection.

Mortality.—The mortality in natural outbreaks varies widely depending on environmental and climatic conditions and the type of secondary infection. The disease may be mild with a low mortality or severe with a high mortality.

Treatment.—Management practices are important in decreasing the severity of an outbreak. Adequate nutrition, proper ventilation, prevention of crowding and protection from inclement weather are essential. Many stress factors appear to increase the severity of CRD. Various field trials on "air sac" infection using antibiotics added to the feed or injected, have shown encouraging results. The results are based on reduction of mortality, fewer culls and improvement of feed conversion.

Recent reports (Crawley and Fahey, 1955, 1957) have indicated that poultry flocks free of PPLO infection may be obtained by the injection of breeding flocks with streptomycin and rearing the chicks away from infected stock. The serological tests are used to identify breeding flocks which are free of PPLO infection.

INFECTIOUS SINUSITIS

This disease was first reported in turkeys in 1905 by Dodd in England and in the United States by Tyzzer (1925). It is characterized principally by inflammation and swelling of the infraorbital sinuses but there may also be involvement of other respiratory organs such as lungs and air sacs.

Cause.—The organism *Mycoplasma gallinarum* that causes chronic respiratory disease in chickens has the same characteristics as PPLO isolated from turkeys and the agents are considered identical.

Occurrence.—Infectious sinusitis has become one of the serious diseases of turkeys and has been reported from all major turkey raising areas of the United States.

Symptoms.—The first symptom usually shown by affected birds is a clear discharge from the nostrils, followed by a

States and Canada Delaplane and Stuart (1943) first described the disease in chickens. It is characterized by nasal discharge respiratory rales slow rate of spread persistency of symptoms moderate decline in egg production and loss in body weight.

Cause—The principal cause of the disease is considered to be an organism belonging to the pleuropneumonia like group (PPLO) *Mycoplasma gallinarum* (Markham and Wong 1952).

Occurrence—Chronic respiratory disease complex has been noted in all the major poultry raising areas of the United States and Canada (Summs 1952).

Symptoms—The first symptom usually shown by affected birds is a nasal discharge followed by a foam or bubbly condition of the eyes. The birds may show a persistent hacking cough and tracheal rales. The respiratory symptoms may persist in the flock for weeks. In living birds egg production is below normal but there is usually no dramatic drop in egg production as seen in Newcastle disease and infectious bronchitis.

Postmortem Appearance—There is accumulation of mucus in the infraorbital sinuses and trachea. The air sac membranes are thickened and there is accumulation of caseous pus in the air sac cavities.

Birds that have a secondary infection show exudate in the heart sac and over the surface of the liver. In field cases of air sac infection these lesions are often considered to be the result of mixed infections due to PPLO bacterial or viral agents.

Diagnosis—Definite diagnosis is dependent upon the isolation of the causative agent *Mycoplasma gallinarum*. In addition serological tests are available. Jungherr has described the use of the rapid whole blood plate serum plate tube test and hemagglutination inhibition test for the detection of recently recovered and carrier birds. Pathological findings are also suggestive of chronic respiratory disease. Because of the interrelationships of various infections in producing field cases of air sac infection a laboratory examination is important in making a differential diagnosis.

Transmission—Van Roekel (1953) and Lahav (1954) reported that egg transmission of the agent may be a primary

vention of crowding, and protection from inclement weather are also to be considered as valuable preventive measures.

The injection of 1 cc. of a 4 per cent solution of silver nitrate into the affected sinuses after withdrawal of the exu-

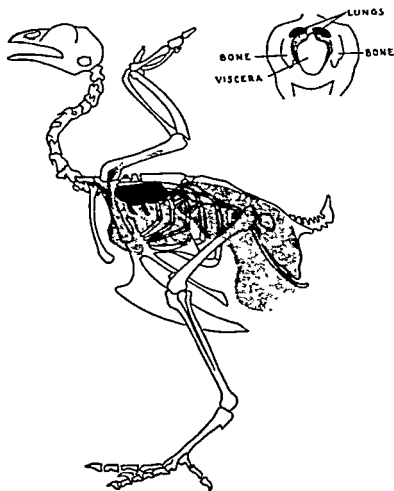


FIG. 35 —Shaded areas show location of air sacs in the chicken and turkey (darkest area represents the lungs). Lesions of the lower respiratory form of infectious sinusitis and chronic respiratory disease may be found in any of these areas. (Courtesy of Minnesota Agricultural Experiment Station)

date by means of a syringe, is recommended by Madsen (1938) as being highly effective in treating the condition. McNeil and Hinshaw (1946) tested several drugs, including the colloidal silver preparations argyrol, protargol, and

foamy or bubbly condition of the eyes. The sinuses gradually become filled with a clear mucoid secretion and the resultant swelling may be so great as to force the eyes to be partially or wholly closed. This secretion may later become cheesy and yellow. Labored breathing is seen in those cases in which pneumonia or bronchitis has developed. Affected birds continue to eat as long as the eyes are not completely closed but there is a gradual loss of flesh.

Postmortem Appearance — Bulging of the infraorbital sinuses which are filled with mucoid or cheesy exudate and loss of flesh are the most frequently observed indications of sinusitis at autopsy. If the lower respiratory system is involved bronchitis and pneumonia may be present. The edges of the eyelids are usually crusted with a brownish dried exudate and are frequently adhered together. Occasionally a cheesy deposit in the air sacs is seen.

Diagnosis — Definite diagnosis is dependent upon the establishment of the presence of the causative agent *Mycoplasma gallinarum* by laboratory procedures but the symptoms and autopsy findings described above are suggestive of sinusitis. Vitamin A deficiency may produce symptoms and lesions which closely resemble those of infectious sinusitis and the similarity of the manifestations of the two conditions should be kept in mind as well as the fact that the two diseases might occur simultaneously in the same bird or flock.

Transmission — Jerstad *et al* (1949) isolated infectious sinusitis agent from fresh turkey eggs and suggested the possibility of egg transmission. Matanov *et al* (1955) in experimentally and naturally infected turkeys clearly demonstrated egg transmission. The causative agent was isolated from infertile eggs, dead and pipped embryos and poults that hatched. Under natural conditions cohabitation plays a part in spreading the disease within a flock.

Mortality — The disease usually runs a chronic course and may exist in a flock for weeks or until marketed. The loss of condition in affected birds and the cost of medication may result in greatly curtailed financial returns.

Treatment — Adequate nutrition is an important preventive measure since it has been shown that a deficiency of vitamin A predisposes to infectious sinusitis. Proper ventilation pre-

to the widespread occurrence of the disease in fowls and their eggs

Occurrence—Paratyphoid infections in birds appear to be widely disseminated, having been reported in many sections of the United States as well as in England and other European countries

Susceptibility.—Infection by *Salmonella* organisms has been reported in chicks, ducklings, ducks, pigeons, turkeys, guinea fowl, canaries, parrots, quail and baby wood ducks. Hinshaw and McNeil (1945) isolated *Salmonella* from snakes, turtles, a Gila monster and an iguana. These findings are of significance in considering the reservoirs of infection and modes of transmission

Symptoms—As has been stated, paratyphoid infections are of principal importance in young birds but older birds are also susceptible. No group of symptoms is diagnostic of the disease in a given species of birds. Affected chicks are dull, listless and weak, there is diarrhea, the vents are smeared, and the birds stay near the hover.

Evans and co-workers (1955) reported that eye infections in chicks involving four variant types of *S. typhimurium* resulted in several cases of blindness.

In young turkeys acutely affected, the poult appears chilled and stay near the hover, while in less acute cases, weakness, unthriftiness, ruffled feathers, dragging wings, and slow, sluggish movements are observed. The birds stand for long periods with eyes closed and the head is either tucked under the wing or hangs forward on the breast. Diarrhea may be present but it is not a constant symptom.

In squabs, Beaudette (1926) noted nervous symptoms, including convulsions, staggering, shaking of the head, twisting of the head and neck, and sometimes loss of the use of the legs. There may also be such manifestations as emaciation, loose greenish feces, inflamed and watery eyes, respiratory disturbance and stiff leg and wing joints. In mature pigeons there may be inability to properly use the wings, which in some cases are drooped, and the birds are slow to fly. Swellings of the joints are frequently observed.

In ducklings, paratyphoid infection is commonly known as "keel disease," the term being derived from the actions of

novocin by injecting them into the sinuses of affected birds but found them less effective than the freshly prepared silver nitrate solution. They also tried two ephedrine solutions containing sulfonamides but found these preparations to be relatively ineffective.

If the exudate in the sinuses has become caseous it can be removed only by surgical means. This is accomplished by incising the bulging skin over the sinuses, expressing the exudate and swabbing the cavity with a good antiseptic, preferably a 4 per cent solution of silver nitrate.

Various studies have been made on the susceptibility of the agent to antibiotics. Streptomycin or dihydrostreptomycin when injected into the sinus is a highly effective form of treatment. Other antibiotics Terramycin* and Aureomycin* have been found effective but are irritating and cause a local reaction. Antibiotics have been used effectively in the feed and water or injected in controlling losses in serious outbreaks of infectious sinusitis.

Because of the danger of egg transmission breeding flocks should be selected from flocks that show no clinical evidence of infectious sinusitis. The same serological tests that have been used on chickens have been applied to turkeys.

SALMONELLA (PARATYPHOID) INFECTIONS

The constantly increasing number of reports of *Salmonella* infections in fowls in addition to pullorum disease and fowl typhoid indicates that the losses caused by this group of organisms are important and of widespread occurrence. In many respects paratyphoid infections are similar to pullorum disease, particularly with reference to transmission through the egg. The infection is primarily a disease of young birds.

Cause—The etiological agents of paratyphoid infections are members of the *Salmonella* group. Over 200 types of the organism have been identified and of this number at least 60 have been found associated with illness in birds. Many of the *Salmonella* organisms are known to be pathogenic for man as well as for other mammals and there is considerable significance from the public health standpoint, to be attached

is unlikely that it will prove of sufficient specificity to warrant its use in the field. The antigenic properties of the many types of *Salmonella* vary and the agglutination test would apparently be of practical value only if the infecting type of organism had been carefully determined, and the testing procedure adapted to the findings.

Dissemination—Paratyphoid infection is readily spread by cohabitation and the addition of newly-hatched, diseased birds may well be an important means of introducing the malady into a flock. There is sound evidence that the infection can be perpetuated in the ovary of the mature fowl, with the cycle of transmission being much the same as it is in pullorum disease. The carrier bird must also be considered a potent means of dissemination, not only by fecal contamination of the feed and water troughs, and yards, but also of the shells of eggs laid by such birds.

The findings of Hinshaw and McNeil (1945) (1947) and of McNeil and Hinshaw (1947) emphasize the importance of such carriers as cats, snakes, and flies, and stress the necessity of the elimination of such reservoirs of infection as a basic factor in any control program.

Mortality—In the reported studies of outbreaks, the death-rate has varied from as low as 10 per cent to the majority of the birds involved.

Treatment—As in many other diseases of fowls, treatment has not proved efficacious. Many drugs have been tried but none has given promise of ability to control the infection. Limited experiments with some of the sulfonamides have been carried out. Hinshaw and McNeil (1946) found that both sulfathiazole and sodium sulfathiazole were ineffective against two outbreaks of acute fowl typhoid. Pomeroy, Fenstermacher, and Roepke (1948) were able to reduce mortality by about 50 per cent in chicks infected with *S. typhimurium*, but sulfathiazole, sulfaguanidine, and sulfadiazine were only about one-half as effective in reducing the losses among poults as compared with chicks when both were infected with *S. typhimurium*.

Aureomycin® and Terramycin® have been used with some success in reducing death losses and furazolidone has proved effective in treating some acute outbreaks, but there is so

the birds in "keeling over" just before dying. The disease has been reported in the United States, Great Britain, and Africa, and it is probable that it exists unrecognized in other countries. Young ducklings from one to three weeks of age are highly susceptible but older birds apparently resist the infection.

The cause of "keel disease" in ducklings is *Salmonella anatum*, first isolated by Rettger in 1918 and subsequently described by Rettger and Scoville in 1920. Affected birds show loss of appetite, watery discharge from the eyes and nose, and weakness. They are sluggish and there is incoordination in their movements. Thirst is usually increased and many of the ducklings soon after drinking, draw themselves up to full height, stagger momentarily, then "keel over" and die.

Affected young guineas show drooping wings, huddling, discharge from the eyes and nose, dribbling of saliva, tossing of the head, clenching of claws, and distention of the infraorbital sinuses.

Postmortem Appearance—The lesions encountered at autopsy are not diagnostic, but among the changes seen are congestion of the liver, kidneys, gall bladder, and myocardium, in birds dying under ten days of age pneumonia is frequently seen and many have retained watery or coagulated yolk-sacs. The liver may be pale yellow and mottled, and white cheesy plugs are commonly found lying loose in ceca of normal size or in those greatly distended. Catarrhal enteritis, especially of the duodenum, is a frequent finding in all species of affected birds. Enlargement of the spleen and the presence of fibrinous exudate on the liver, spleen, and lungs are sometimes noted.

Diagnosis—The symptoms and postmortem lesions noted in *Salmonella* infection cannot be relied upon as diagnostic because of their marked similarity to those found in other diseases and also because of their inconstancy. The isolation and positive identification of the causative organism in a properly equipped laboratory is the most reliable means of diagnosing the condition.

The agglutination test has been studied extensively as a means of diagnosis, but in the light of present knowledge it

(1948) ascertained the ability of paratyphoid germs to penetrate the shells of eggs and contaminate the contents.

In their studies at the New York Salmonella Center, Seligman, Saphra, and Wassermann (1946) found that the number of human carriers is large; of all stool cultures submitted, 19.5 per cent were involved, predominantly those from children.

It is evident that the cross-infectiousness of many of the *Salmonella* organisms for man and fowls, as well as for other animals, poses a serious public health problem which merits intensive study.

ARIZONA (PARACOLON) INFECTIONS

The Arizona group of bacteria is closely related to the *Salmonella* group. Members of this group have been referred to as paracolons but Arizona group is preferred terminology. The organisms produce a disease in turkeys and chickens similar to *Salmonella* infections and are of considerable economic importance to the turkey industry.

Cause.—The etiological agents of Arizona infections are organisms that have been identified by serological and biochemical tests and 96 serotypes have been isolated from fowl, mammals, reptiles and man.

Occurrence.—The Arizona organisms in birds appear to be disseminated in most of the turkey raising areas of the United States.

Susceptibility.—Infection by Arizona organisms has been reported in chicks by Lewis and Hitchner (1936), Edwards (1947) and in poults as well as snakes by Hinshaw and McNeil (1944).

Symptoms.—Arizona infections are of principal importance in young birds but older birds may remain as carriers.

There are no diagnostic symptoms of this group that will differentiate it from pullorum disease, fowl typhoid and paratyphoid infections. In some outbreaks blindness and nervous symptoms may be noted.

Postmortem Appearance.—The lesions encountered at autopsy are not diagnostic but are suggestive of the *Salmonella* or Arizona group. White cheesy plugs are commonly found

for no treatment which can be given an unqualified recommendation

Control—The most effective method of control undoubtedly is the establishment and maintenance of a flock free of the infection. In the experience of Cherrington Gildow and Moore (1937) cleanliness and sanitation of incubators and brooders including formaldehyde fumigation of incubators did not reduce mortality.

The present status of the agglutination test renders its use unsuitable as a practical control measure unless careful typing of the infecting organisms and complete supervision of the tests are done by well qualified persons. It is to be hoped that further studies of the agglutination test may result in its development to the point where it can be relied upon for general field use for the detection of carrier fowls.

Rigid sanitary conditions and practices must be carried out in attempts to control paratyphoid infections. As stated previously the importance of controlling spread of the infection by such reservoirs as cats snakes flies rats and lizards should be recognized.

Those engaged in any phase of poultry husbandry should be guided in handling paratyphoid infections by competent veterinarians who have access to complete laboratory facilities where diagnosis can be quickly made enabling indicated control measures to be established.

Fowl Paratyphoid in Man—It is well known that many of the *Salmonella* organisms which infect fowls are also capable of causing disease in humans. Clarenburg (1939) states that a large number of cases of food poisoning in humans in the Netherlands have been caused by the consumption of contaminated duck eggs insufficiently cooked. He also calls attention to the possibility of the organism being present both in the egg and on the shell and both possibilities have subsequently been proved to be facts. Schneider (1946) reported the isolation from powdered whole egg of 17 types of *Salmonella* some of which have been incriminated as causing disease in humans. He also reported the presence of 3 types in frozen whole eggs. Cantor and McFarlane (1948) found *Salmonella* organisms on and in chicken eggs and Gregory

inflammation of the mucous membranes of the upper respiratory tract, in other words a typical coryza

Once introduced into a flock, it spreads rapidly and may involve nearly every bird in the flock within a very short time. It occurs most frequently in damp, cloudy weather when there are sudden and marked changes in temperature. Crowding and lack of ventilation are predisposing factors.

A diagnosis of infectious coryza is indicated with any respiratory disease which affects a rather high percentage of the flock, which persists for several weeks, and which has a nasal discharge as a constant symptom. This is especially true if pullets on a given farm are affected year after year.

Cause—The complete etiology of infectious coryza probably has not yet been determined, but at least two distinct types are recognized. One is characterized by rapid onset—nasal discharge appearing on the first or second day after artificial inoculation. This type is of relatively short duration in the laboratory, but usually runs a longer course when produced by natural infection or by inoculation with nasal exudate. The other type is characterized by slow onset, the nasal discharge appearing two to four weeks after inoculation. This type is likely to persist for several weeks.

Invariably associated with the first type, but apparently never with the second, is a pleomorphic, hemophilic, Gram-negative bacterium known as *Hemophilus gallinarum*. Working with the second type, Nelson (1936) isolated a specific causative agent in the form of minute bodies which he called "cocccobacilliform bodies" and which were capable of producing the infection. Later he showed that coryza of rapid onset and short duration is produced solely by *H. gallinarum*, that coryza of slow onset and long duration is produced by the cocccobacilliform bodies, and that a third type of rapid onset and long duration occurs when both agents are present. Type III was consistently produced by injecting a mixture of the two agents (Nelson, 1938).

Adler and Yamamoto (1956) repeated Nelson's studies with *Hemophilus gallinarum* and obtained essentially the same results which he described in 1936 and 1938.

Symptoms—The first symptom observed is a thin, serous discharge from the nostrils. This soon becomes thick and

in the ceca. The eyes may appear cloudy in birds that are blind.

Diagnosis—The symptoms and lesions are so similar to *Salmonella* infections that the diagnosis is dependent on the isolation and identification of the causative organism in a properly equipped laboratory.

Dissemination—The carrier bird must be considered a potent means of dissemination through infected eggs as well as by fecal contamination of feed, water, litter and yards. The cycle of infection is similar to pullorum disease and fowl typhoid. Other carriers such as snakes and mammals may be the source of the infection in poultry.

Mortality—In experimental infections the mortality is dependent on the age of the poult or chick at the time of exposure. If poult are exposed at time of hatching or before started on feed and water the losses may be high, 50 to 100 per cent. In natural outbreaks the mortality may vary from 0 or 10 per cent to 60 per cent.

Treatment—Very little experimental work has been done with the newer antibiotics and chemotherapeutic agents on the Arizona group. Furazolidone at 100–200 grams per ton (Pomero, 1957) has been found effective in reducing the mortality in experimentally infected poult.

Control—The most effective method of control is the establishment and maintenance of a flock free of the infection. The application of the agglutination test using the specific serotype involved in an outbreak as an antigen may be considered in locating infected breeding flocks. Proven infected breeding flocks should not be used as a hatchery supply flock.

Rigid sanitary conditions and practices must be carried out to prevent the introduction of the infection from outside sources.

Advice of the State diagnostic laboratories and animal disease control officials should be followed in developing a control program.

INFECTIOUS CORYZA

This disease which has long been known among practical poultrymen as roup, or simply as colds, is a severe catarrhal

Borstein and Samberg (1955) reported that intramuscular injection of 0.2 gm. of dihydrostreptomycin sulfate in hens affected with infectious coryza brought about a remarkable clinical recovery within forty-eight hours and a prompt resumption in egg production.

Fowls which recover from coryza become carriers and are the most common source of new outbreaks. Unless pullet stock is completely segregated from any such carriers, the disease is almost certain to recur year after year on the same farm. Prevention can be accomplished only by disposing of all fowls after an outbreak, or by complete and permanent separation of pullets from older stock. One definite advantage of the so-called all-pullet flock, with 100 per cent replacement of layers each year, is complete freedom from infectious coryza.

ERYSIPELAS INFECTION

The occurrence of a disease in birds caused by the organism of swine erysipelas has been reported with increasing frequency and heavy losses have occurred in some outbreaks, particularly in young turkeys. The condition is usually acute or subacute in character and it assumes importance epidemiologically because several mammals, including swine, sheep, and man are susceptible.

Cause.—Erysipelas in fowls is caused by the germ *Erysipelothrix rhusiopathiae*. It is a fairly resistant organism and is capable of surviving drying and other adverse conditions for considerable periods of time.

Occurrence.—Erysipelas infection occurs frequently in the United States and has also been reported from other countries.

Susceptibility.—Infection with *Erysipelothrix rhusiopathiae* has been reported in several species of birds including turkeys, chickens, pigeons, ducks, coots, thrushes, parrots, quail, pheasants, parakeets, and peacocks. The same organism has also been found in several species of mammals, particularly swine, sheep, and humans.

The severest losses occur in young birds, especially poults, and is far more prevalent in males than in females. Madsen

sticks, with an offensive odor and has a tendency to dry in yellowish crusts around the nasal openings. As the inflammation extends the adjacent sinuses become filled with mucus. This is unable to drain away and changes to a rather dry, cheesy form which accumulates in such quantities as to cause prominent bulging about the eyes. If the deeper air passages are involved breathing is accompanied by rattling noises. Affected fowls sit quietly with feathers ruffled, have little or no appetite, shake their heads frequently in an effort to dislodge the mucus, and lose strength rapidly. Yellowish, soft patches may form in the mouth and these add to the difficulties of the fowl in breathing. They also form the basis upon which some writers have attempted to distinguish between a 'mouth type' and an 'eye type' of the disease.

Postmortem Appearance—There are no characteristic lesions other than those described as symptoms. In distinguishing infectious coryza from the diphtheritic type of fowl pox it is well to remember that the yellowish patches in the throat are easily removed while in pox they are very adherent. Of course it is also true that fowl pox can usually be identified by the presence of characteristic pox nodules in at least some specimens.

Treatment and Prevention—Individual treatment of various sorts has been shown to be effective, but it is usually too costly to be practical. Flock treatment has not been too effective. Heiman (1943) reported that feeding $\frac{1}{4}$ gram of sulfathiazole per ounce of feed (about 7 ounces per 100 pounds) kept cold infections to a maximum of 20 per cent in contrast to 90 per cent for similar groups of young chickens in adjoining battery compartments but untreated. When sulfathiazole was discontinued, the percentage of colds increased to 80 within seven days. Growth was significantly better in the sulfa treated groups.

More recently McKay (1948) reported that severe and persistent outbreaks on five large poultry farms involving 25 000 birds were treated with rapid and marked success by iodizing the drinking water to a concentration of 1 part of 'free' iodine to 20 000 parts of water. This was accomplished by adding Lugol's solution at the rate of 1 teaspoonful to each gallon of water.

dependent upon isolation and identification of the causative organism in the laboratory. Because of the infectiousness of *E. rhusiopathiæ* for other species of animals, a competent veterinarian should be consulted when the disease is suspected.

Dissemination.—The means by which the infection is spread are not definitely known. In the outbreak reported by Madsen (1937) one of the affected flocks was so located



FIG 36.—Turkey showing swollen snood as a result of erysipelas infection
(Courtesy of Minnesota Agricultural Experiment Station)

that the manure from sheep corrals washed down during heavy rains and since erysipelas is not uncommon in sheep, it is possible that diseased animals in the band were the source of the infection. Affected swine might well be the source of the causative organisms. Stiles (1946) states that purchased, infected pigs were the probable source of the trouble in a flock of turkeys in which the losses were significant. Because many of them are susceptible, wild birds might also serve as agents of transmission.

(1937) reported an outbreak in which over 92 per cent of the dead birds were males, and Rosenwald and Dickinson (1939) reported that 82.1 per cent of the turkeys involved in several outbreaks studied by them were males. They also reported that most of the cases occurred in October, November, and December. Stiles (1946) noted that the losses were 10 times greater in males than in females in an outbreak observed in Colorado.

Symptoms—Affected poults are listless, weak, and indifferent to approach. There is usually cyanosis of the skin of the head, the wings droop, the feathers are ruffled, and a greenish or a yellowish diarrhea is often observed. Loss of appetite occurs in severe cases but it is not so marked as might be expected. In some affected birds dyspnea is also observed. In general the symptoms are such that they might be confused with those of blackhead.

Postmortem Appearance—At autopsy numerous lesions are observed but these vary from case to case and cannot be considered as characteristic. In most cases in poults, hemorrhages of varying size are present in the pectoral, thoracic and abdominal muscles, over the ribs, and occasionally in the region of the thigh. Hemorrhages may also be seen in the pericardium, epicardium, pleurae, and peritoneum. Thick mucus is usually found in the nasal cavity. The liver is regularly involved, showing enlargement, congestion, and friability, while mottling may be noted in some cases. Rosenwald and Dickinson (1942) state that the most pathognomonic lesion in turkeys is a turgid, reddish purple caruncle, but this change is also occasionally observed in other diseases, especially cholera. In the majority of cases, inflammation of the anterior part of the intestine is present and in some cases the large bowel is similarly involved. The spleen usually presents alterations which include friability and congestion, and similar involvement of the lungs and kidneys is occasionally observed.

Diagnosis—The similarity of the symptoms of blackhead and of erysipelas infection may render differentiation difficult. Paratyphoid infection might also conceivably be confused with erysipelas. As is the case with many other diseases of the fowl positive diagnosis of the disease is

Removal of healthy appearing birds to new quarters, whenever practical, aids in reducing the spread of the disease. Care should be used in purchasing additions to the flock, to make sure that they come from sources free of the infection.

BOTULISM

Botulism in fowls is an acute or subacute disease which is characterized by marked weakness and prostration. It is commonly called "limberneck" because in many cases affected fowls lose control of the neck muscles, and are unable to hold up their heads.

Cause—The cause of the disease is a powerful toxin, produced by the microorganism *Clostridium botulinum*. This toxin is one of the most poisonous substances known and minute amounts of it are highly fatal to susceptible animals. Only types A and C of the organism produce toxins affecting fowls. The organism itself is only moderately resistant to disinfectants and heat, but it produces spores or "seeds" which are highly resistant to outside influences as well as to disinfectants. The spores of some strains resist boiling for as long as three and a half hours, while those of other strains are destroyed by this treatment in a few minutes. The spores themselves, as well as the organisms, are non-toxic.

Clostridium botulinum is commonly found in the soil and no section of the country appears to be wholly free of it. The organism has been incriminated in many outbreaks of so-called "food poisoning" of man and it is not infrequently found in canned foods, especially spinach, corn and beans. A relative absence of oxygen is necessary for the organism to produce its toxin, and such an environment is readily provided in canned foods and in the carcasses of dead animals. Low, wet places favor survival of spores and, under favorable conditions, the organisms resulting from such spores produce toxin. Fly maggots found in carcasses of animals dead of botulism often contain enough of the toxin to be fatal to birds devouring them. Quortrup and Sudheimer (1943) made the interesting observation that there apparently is a strong symbiotic relationship between *Cl. botulinum* and *Pseudomonas aeruginosa*. The latter organism has great

Mortality—Heavy losses have been reported in some outbreaks of erysipelas infection in birds, while in other cases, the deaths have not been numerous.

Treatment—The treatment of erysipelas in fowls with drugs has not been promising. Sulfanilamide was tried by Ro-enwald and Dickinson (1941) and by Lindenmayer and Hamilton (1942) without satisfactory results.

Vaccines, prepared with added saponin did not have practical value and culture filtrates likewise were not effective at immunizing agents (Ro-enwald and Dickinson, 1941). These workers also found commercial anti-swine erysipelas serum to have no practical value in either treatment or prophylaxis of the disease. Bullis and Clarke (1938) however reported that the intraperitoneal injection of anti-swine erysipelas serum checked the progress of the disease in one flock under their observation, and Grey (1947) was able to reduce mortality in experimental birds to 50 per cent while 100 per cent of the controls died by use of the anti-serum.

Penicillin gave inconclusive results for Stiles (1946) but Grey reduced mortality to 10 per cent by its use in experimental turkeys. Streptomycin was used by Grey in single doses in turkeys and provided complete protection as compared with a death loss of 80 per cent in untreated fowls.

More recently erysipelas bacterin and some of the antibiotics have proved useful under field conditions. Further research may lead to an effective method of control.

Control—Fowls, particularly turkeys, should not be allowed to come in contact with sheep or swine, nor should they be allowed to run over ground on which these animals have ranged. Ailing birds should be removed and destroyed by burning and the apparently well fowls moved to uncontaminated premises if at all possible. The need for cleaning and disinfecting the premises and utensils is apparent. Cleaning and disinfection of utensils and houses should be carried out frequently. It is especially important to remove and burn the droppings daily during an outbreak. All low wet places should be abolished, and particular attention paid to placing the water and feed troughs so that it is impossible for the birds to contaminate them with their feces.

small amounts of the toxin may induce only moderate symptoms including leg weakness and dullness and ending in recovery. In the fatally poisoned birds, death may ensue rather rapidly, or the birds may linger for several hours before they finally succumb.

That botulism may, however, at times be accompanied by atypical symptoms is shown by the report of Coburn and Quortrup (1938). In affected turkeys they observed slight cyanosis of the heads, conjunctivitis in one bird, as well as the common symptoms of general weakness, leg paralysis, and

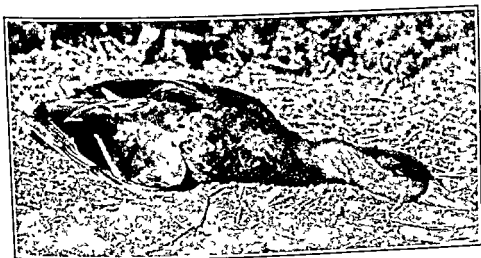


FIG 37.—A Mallard duck affected with botulism (commonly called limber-neck) It is caused by a powerful toxin produced by the organism, *Clostridium botulinum* (From the U. S. Department of Agriculture)

inability to stand. These authors consider paralysis of the nictitating membrane to be pathognomonic of botulism but this symptom was observed in only a few cases out of many affected birds. The toxin of *Cl. botulinum* type C, was found in the water of a stagnant pool to which the turkeys had access, and the organism was readily isolated from the surrounding soil.

Postmortem Appearance.—Botulism produces no diagnostic changes in the organs of a poisoned fowl. Varying degrees of inflammation may be noted in the lining of the intestine but this is not a constant finding. A great deal of emphasis

oxygen-consuming, alkali producing qualities and thus may readily produce conditions under which *Cl botulinum* thrives. The presence of such an organism as *Ps aeruginosa* readily explains the occurrence of botulism in areas where it would not normally be expected as for instance, in water having a depth in excess of one foot but containing masses of decomposing vegetation.

Occurrence—Outbreaks of botulism in fowls have been reported from widespread localities in the United States. Kalmbach (1935) states that the disease exists in wild fowl from well north of the Canadian border south to Arizona and New Mexico, and from southwestern Minnesota west to California. Sixty-nine species of wild birds embracing 21 families, besides many shore birds and waterfowl, several passerine birds, gulls, terns, hawks, herons, cormorants, pelicans and grebes are listed by Kalmbach (1939) as being susceptible to botulism. In extensive field observations however, this author reports never having seen a case of botulism in turkey vultures. He showed by experimentation that these birds resist large doses of the toxin and that their blood serum possesses inactivating substances for the toxin, and therefore it must be concluded that the turkey vulture is a naturally resistant bird. Pullar (1934) has observed deaths from botulism in cormorants, plover, ducks, pelicans, ibis, and hawks.

Fowls are highly susceptible to the A and C toxins and within the last few years large losses in wild ducks have been traced to the ingestion of material containing type C toxin. The disease is more prevalent during warm weather because of the favorable conditions for toxin production, but the feeding of spoiled foods may, of course, produce the disease at any time.

Symptoms—The first noticeable symptoms in poisoned fowls are dullness, sleepiness and leg weakness. The affected birds are soon unable to stand, and the neck and wing muscles are paralyzed, causing the head and wings to be rested on the ground. Coma follows the dullness, and the poisoned birds lie sprawled about in various positions on the ground, appearing to be lifeless. If the birds are handled, the feathers are found to be loose and easily plucked. Ingestion of very

mottled, with many brownish or gray foci, and at death was friable and pale, the kidneys were degenerated and pale, the ovary was always non-functional and degenerated, with the ova becoming flaccid and dirty yellow in color. There was a necrotic enteritis, with irregular elevated areas of cell infiltration in the duodenum. In three of four naturally infected flocks, egg yield was decreased. The agglutination test was found to be fairly reliable as a means of detecting the disease, although a negative test did not preclude infection, and in most of the experimental fowls the agglutination tests were negative in the last stages of the disease. Emmel (1930a) found turkeys, pheasants, ducks and geese susceptible to infection when fed massive doses of the organism and the same author (1930b) found 16.5 per cent of a flock of 90 chickens to give positive reactions to the agglutination test. Many of the affected birds were in poor physical condition and egg production was greatly curtailed.

Other workers have studied the effect of the abortion organism upon fowls and found it to possess little pathogenicity for them. McNutt and Purwin (1930a) found that but a small percentage of the birds in 20 flocks gave positive agglutination tests, with no symptoms being shown by the reactors. The same authors (1930b) attempted to infect birds by both feeding and injection, but none of the birds showed symptoms and no deaths occurred. In 69 flocks having a total of over 10,000 fowls, less than 2 per cent showed evidence of infection, no flock contained more than 12 per cent reactors, and none of the birds evinced any symptoms of illness. Strange and Berch (1931) failed to induce clinical evidence of disease in 32 chickens by injection and feeding and concluded that chickens are not ordinarily susceptible to infection by the abortion organism. In a later report McNutt and Purwin (1932) found that feeding of *Brucella* organisms to laying pullets was followed by a temporary, slight decrease in egg production, but that no effects were produced in ten-day chicks. Gilman and Brunett (1931) found but a small percentage of the birds in four flocks of chickens to give positive agglutination reactions but concluded that evidence points to the presence of natural infection in farm flocks.

has been placed upon finding the larvæ (maggots) in the crops of affected fowls, but their presence is by no means diagnostic, since they are prevalent in many materials in which no botulinum toxin is present. The symptoms displayed by affected fowls are of far greater aid in diagnosing botulism than are the inconstant lesions found at autopsy. In case spoiled foods have been thrown out to the birds, followed by the symptoms mentioned botulism should be suspected.

Mortality—The mortality is dependent upon the number of fowls ingesting fatal doses of the toxin. It may vary from a few deaths to a catastrophe involving the entire flock.

Treatment and Prevention.—Treatment of visibly affected fowls is of little value. Epsom salts, given in the drinking water at the rate of 1 pound to each 100 birds, aids in flushing the digestive tract. The administration of botulinus antitoxin is an effective means of preventing the disease in exposed birds, but if symptoms are being manifested it is usually too late to derive any benefit from the use of antitoxin.

If botulism is suspected, careful search should be made for any possible source of the toxin. If decomposing carcasses of any kind are accessible to the fowls they should be removed and burned. Suspected feed should be replaced by clean, wholesome materials. As a matter of prevention, the feeding of spoiled canned goods or tainted food of any kind is to be discouraged because of the disastrous results which may follow.

BRUCELLA ABORTUS INFECTION

Although natural infection of fowls by the abortion organism undoubtedly occurs, the disease is apparently of little economic significance. Emmel and Huddleson (1929) fed naturally infected milk, portions of an aborted fetus, and cultures to fowls and produced infection. The symptoms were loss of production, increasing paleness about the head, diarrhea and emaciation. The birds became very weak and often showed paralysis. The postmortem changes varied, depending upon the course of the disease, but the spleen was at first enlarged, later shrunken, the liver was pale and

articular surfaces of the bones, but the tissues around the joints showed marked inflammatory changes. The author isolated an organism having the characteristics of *Staphylococcus aureus*.

Fahey (1954) described an outbreak of staphylococcal arthritis in turkey poults in which over 80 per cent of the birds were infected. Injection of an antibiotic mixture containing penicillin, dihydrostreptomycin and Terramycin, followed by the use of Terramycin in the feed resulted in complete disappearance of symptoms and full recovery of the poults.

Jungherr and Plastridge (1941) have recorded an outbreak of staphylococcal arthritis in a group of young cockerels. Cultures of staphylococci, both pathogenic and non-pathogenic were isolated from affected birds. The cockerels had been fitted with an anti-picking device which required insertion through the nostrils and the authors consider it possible that the resulting wounds may have been the portal of entry of the causative organisms. Swellings, unilateral and bilateral, of the tibiotarsal joints were present and the affected birds were lame, moving with a painful gait. In chronic cases kept for several weeks, the swellings became firmer and more localized, and the material in the swellings was purulent or caseous, whereas in the acute cases the fluid in the swollen joint was serous in character. Some cases were observed in which dark colored crusts or scabs were present on the comb. The losses were severe with 60 out of 100 cockerels succumbing.

Losses in two-month-old fowls, amounting to 20 per cent of a flock of 200 and caused by *Staphylococcus citreus* were described by Van Ness (1946). The affected birds showed cysts of the keel and arthritis, while in dead fowls there were keel cysts, joint involvement, and cyanosis of the liver with focal necrosis. Cysts of the keel were the primary lesions followed by arthritis and in some cases, septicemia. The keel lesions were believed by the author to have been caused by injury from jagged points on the wire fence with resultant introduction of the infection. Mortality was most severe in bad weather.

The experimental evidence on brucellosis in fowls is as yet rather meager, and there appears to be some difference of opinion as to the importance of the disease in birds. Further studies are necessary to gain evidence regarding the extent and importance of the disease.

STAPHYLOCOCCIC INFECTIONS

Staphylococci are not infrequently found associated with disease conditions in fowls among them being omphalitis, arthritis, vesicular dermatitis, and "bumblefoot."

Arthritis and Periostitis in Pheasants—Hole and Purchase (1931) describe this disease condition in pheasants, caused by *Staphylococcus aureus*. Illness was first noted in six- to ten-week-old pheasants, but no cases occurred in birds after the fourteenth week. The symptoms included lameness, hobbling gait, and frequent resting on the hocks, usually one or more joints were swollen (especially the hock) and occasionally there was a small corn-like spot on the pad of the foot. Postmortem changes were usually associated with the joints, in which there was erosion of the articular surfaces, with cheesy pus present. In other cases there was pus in the tendon sheaths and in the substance of the bone. Involvement of the wing joints was fairly frequent. The authors believed that the infection gained entrance through wounds, with thistles being strongly suspected as the means of breaking the skin. Vaccines were ineffective in controlling the infection.

Staphylococcal Arthritis—This condition was described by Jungherr (1933) as occurring in 25 turkeys in a flock of 450 seven months-old birds. Some of the affected turkeys recovered while others became emaciated and died. Lameness was the predominant symptom. Some of the birds were droopy and emaciated, but most of those affected were in good condition. Two of the birds were able to stand, but when made to move did so with a peculiar hopping motion. Others kept the legs in peculiar positions, and one rested on its hocks most of the time. At autopsy, swellings of the hock and foot joints were noted, some of which contained cheesy material. There appeared to be no involvement of the

There were no lesions in the internal organs when they were examined *postmortem*.

The original outbreak lasted about one month, but in birds which had seemingly recovered, new lesions appeared. The condition apparently was infectious and cultures of an unpigmented staphylococcus were isolated from affected fowls. The author was able to reproduce the disease by using the staphylococcus isolated from diseased birds, as well as with one obtained from a case of human impetigo.

Another condition, also termed vesicular dermatitis or "sod disease," has been described by Newsom and Feldman (1920). It is primarily a disease of chickens under one month of age, although it also occurred in mature hens, and was prevalent in May, June and July. The cause was not definitely established.

The disease is characterized by formation of blisters between the toes, and the entire foot is swollen and tender. Within a few days the blisters break and are replaced by thick, heavy scabs which enlarge and become rigid. In severe cases a joint or even an entire toe sloughs. Mortality ranges from 20 to 90 per cent, but foot distortion renders survivors practically worthless. No effective treatment is known, but prevention was accomplished by keeping young chickens away from virgin sod.

Bumblefoot.—An abscess of the foot is commonly called bumblefoot. The primary cause is a bruise or cut on the bottom of the foot through which organisms, including *Staphylococcus aureus*, gain entrance. (See Chapter 4.)

Omphalitis in Baby Chicks and Turkeys.—Inflammation of the navel in baby chicks has been described by Volkmar (1929) and by Brandly (1932). Volkmar concludes that after the chicks hatch, the navel fails to close properly following the drawing of the yolk-sac into the abdominal cavity, and infection thus gains entrance. The affected chicks observed by him were drowsy and indifferent and appeared as puffed-up balls. Diarrhea was sometimes present and death occurred in three to ten days. A small scab was formed over the navel which when removed left an ulcer-like area. The yolk-sacs were always unabsorbed and somewhat dried out. The liver was pale yellow in color, and the gall-bladder greatly distended with viscous bile.

Vesicular Dermatitis.—Under this designation Hoffman (1939) described a condition in a flock of 2,600 hens which was characterized by the appearance of vesicles in the early stage and later by amber-colored scabs on the comb, wattles, face, feet, and shank. Egg production was severely lowered and the mortality was about 10 per cent. Pullets on the same farm did not develop the disease.

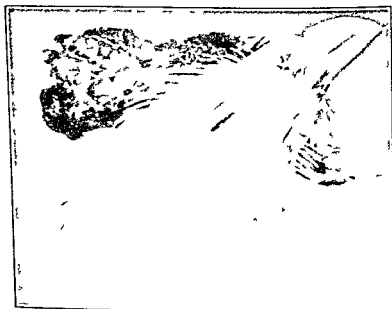


FIG 38.—Vesicular dermatitis. Scabs on comb, wattles, eyelids and around the beak. (Hoffman, courtesy of Jour. Am. Vet. Med. Assn.)

The onset was rapid with vesicles appearing on the comb, wattles, and other unfeathered portions of the head, hocks, and feet. The contents of these blebs were milky, sometimes greenish in color. The vesicles were soon replaced by crusts or scabs, and in many birds the eyelids were stuck together. As the disease progressed, new lesions appeared and in some cases involved the entire skin from hock to feet with a resultant loosening of the scaly portion. The scabs were firmly attached and persisted for at least three weeks.

and later in Germany by Dammann and Manegold (1905) and by Greve (1908). The acute form of streptococcosis is termed "apoplectiform septicemia" and the subacute or chronic form is designated "sleeping sickness." Hudson (1933) reported an outbreak of the acute type in chickens and Volkmann (1932) described streptococcosis in turkeys.

Cause—The cause of the condition is *Streptococcus gallinarum*.

Symptoms—Acutely affected fowls often show no visible symptoms before death. In the less acute cases the affected birds appear sleepy and depressed and may sit motionless for some time. The head is drawn back toward the body and the feathers are ruffled. There may be a slight discharge from the eyes, and there is paleness of the combs and wattles. Very little food or water is taken, and if the disease is prolonged there is marked emaciation.

Postmortem Appearance—The lesions observed at autopsy somewhat resemble those seen in fowl cholera. There may be reddish discoloration of the muscles in various parts of the body although the combs and wattles are usually pale. The liver is enlarged and friable and may show small, grayish necrotic spots. The spleen and kidneys are swollen and there is catarrhal inflammation of the intestines. There are congested areas in the lungs and small hemorrhages are often seen in these organs. The pericardial fluid may be either pale or reddish in color and the heart is full and distended.

Transmission—Hudson (1933) regarded the portal of entry of the infection to be by way of the nasal cavity and not by way of the alimentary tract. He demonstrated that some birds resist artificial inoculation, but become carriers from which the organism can be isolated. Introduction of the infection into a flock might thus readily occur if apparently well carrier birds are added to a flock.

Treatment and Control—No reports of successful medicinal treatment of this condition have been made. Control measures should include precaution against the introduction of affected birds and the institution of strict sanitary measures.

Other Streptococcic Infections—Under the name of *idiopathic streptococcic peritonitis* in poultry, Kernkamp (1927)

Numerous organisms, including *S. pullorum*, were isolated and the author believes that the increased bacterial content of the air in the incubator at hatching time is a factor in the production of the disease.

Brandly noted that most of the losses from omphalitis occurred within seventy-two hours after hatching. The course of the disease was always rapid with death taking place in from two to eight hours, being preceded by prostration and coma. At autopsy he found a parboiled red color of the abdominal skin and muscles with some edema and gas formation. In the abdominal cavity there was a red, serous exudate and the liver and kidneys were pale and swollen. The contents of the yolk-sac were more fluid than normal, and there was a marked putrefactive odor. Numerous organisms were isolated. Brandly concludes that the condition may be related to the influence of high relative humidity in preventing normal enclosure of the yolk sac within the body cavity.

In an outbreak of omphalitis in poults, Williams and Dames (1942) concluded that the causative agent was *Staphylococcus aureus*. Losses ran as high as 30 per cent within seventy-two hours after hatching and the survivors were light and unthrifty with additional losses occurring subsequently. The symptoms were essentially the same as described above. The incubators were operated with a person working inside them, and the authors believe the organism was introduced into the machine by the operator. It is interesting to note that a man herding turkeys on the place developed lesions on the face from which an organism similar to that found in the poults was isolated. The disease did not recur the next hatching season when the operator worked outside the incubators which had been fumigated with formalin in the meantime.

Cleanliness of incubators especially at hatching time should be stressed as a possible means of preventing the disease.

STREPTOCOCCIC INFECTIONS

Apoplectic Septicemia.—This condition was first described in the United States by Norgaard and Mohler in 1902

affected ducks developed a more chronic form of the disease but died later

The changes noted at autopsy were those of a rapid septicemia and included hemorrhages on the spleen, liver and heart, and a fibrinous exudate covering the liver. The pericardium was adherent to the heart wall and there was edema of the heart. The spleen was mottled with brown and white but never enlarged. The kidneys were congested and there was a severe hemorrhagic inflammation of the small intestine. The lungs were highly congested, sometimes pneumonic, with numerous hemorrhages in the membranes lining the bronchi, trachea and turbinates. The authors were able to reproduce the disease readily. No treatment was suggested although the possible value of a vaccine is mentioned.

Bruner and Fabricant (1954) studied the organism in a series of careful tests and concluded that it should be designated as *Moraxella anatipestifer*.

ULCERATIVE ENTERITIS

This disease is frequently encountered in game-breeding establishments and in many cases causes enormous losses. Quail, pheasants, and grouse are highly susceptible and the disease has been found in wild turkey poults, partridges, and occasionally in baby chicks.

Cause —The causative agent was at one time believed to be *L. coli* but Shillinger and Morley (1934) were unable to definitely determine the etiological factor during their studies. Bass (1939) has isolated a "quail disease bacillus" from affected birds and has been able to transmit the disease to susceptible quail by means of it. He describes the organism as a Gram-negative, non-sporulating, strictly anaerobic bacillus.

Symptoms —The malady is acute in character and affected birds sometimes die without manifesting symptoms. As a rule, however, dullness, inappetence, ruffled feathers and diarrhea are observed.

Postmortem Appearance —The principal lesions are to be found in the intestines, lungs and liver. The intestines, par-

described a disease caused by *Streptococcus pyogenes*. The affected birds huddled together with heads drawn back and the eyes partly closed. The feathers were ruffled and lusterless and the feces soft and of a yellowish brownish or greenish color. Sick birds sat on the ground and made no attempt to fly to the perch. The disease ended with prostration and death. At autopsy the visceral surface of the peritoneum and ventral surface of the liver were covered with varying amounts of fibrinous exudate which was soft and easily detached. The liver was slightly enlarged and friable and there were many tiny hemorrhages on its surface. The kidneys were slightly congested and easily torn. The greater number of cases were secondary to other diseases and females were more susceptible than males.

Edwards and Hull (1937) reported finding hemolytic streptococci to be the cause of chronic peritonitis and salpingitis in hens. The affected birds lost weight, became pale and finally died. The appetites were retained and the hens were alert until shortly before death. In addition to peritonitis the oviducts of the affected birds were found at autopsy to be inflamed, enlarged and filled with concretions and purulent exudate.

MORAXELLA ANATIPESTIFER IN YOUNG DUCKS

Hendrickson and Hilbert (1932) described a then new and serious disease affecting young ducks in which the causative organism isolated by them was given the name *Pfeifferella anatipestifer*. Hilbert and Witter (1936) subsequently reported the infection in semi-wild black ducks from two days to twelve weeks of age in which the losses were severe. The disease as originally described by Hendrickson and Hilbert first occurred in birds from seven to ten weeks of age but later affected three-weeks-old ducks. The mortality rate was high with death ensuing within six to twelve hours after the first symptoms were noticed. The symptoms included depression, sleepiness, ruffling of feathers, greenish coloration of feces, jerking of the head, serous discharge from the eyes and weakness followed by prostration. A few

(1945) to be valueless in treating or preventing the disease. Bass (1941) was able to induce a high degree of immunity against experimental infection by injecting intramuscularly small doses of a bacterin prepared from cultures of the bacillus. Further development of the possibilities of preventive vaccination are indicated in view of the promising experimental results thus far reported.

Kirkpatrick and co-workers (1950, 1952, 1953) have reported encouraging results from the use of streptomycin in the control of both experimental and spontaneous ulcerative enteritis in quail.

Scrupulous cleanliness about the pens in which quail and other susceptible birds are reared; provision of an adequate diet to minimize coprophagy; and the immediate segregation of unaffected birds are indicated as control measures. The need for exercising all known means for suppressing the disease is obvious for there are indications that the condition may profoundly affect the population density of wild quail in their natural habitat.

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ticularly the lower part of the ileum and to some extent the ceca, are studded with numerous lentil-shaped ulcers, which in some cases perforate the gut. The duodenum is sometimes inflamed and purplish red in color. Congestion is always present in the lungs and in some instances areas of solidification are noted. Occasionally small necrotic foci are found on the surface of the liver.

Diagnosis—The work of Bass (1939) (1941) appears to have established the cause of ulcerative enteritis to be the organism described by him. Verification of the relationship of the organism to the disease would necessitate the isolation of the bacillus by laboratory procedures for a positive diagnosis. The appearance of the described symptoms and lesions, particularly in young birds, should, however, point strongly to the presence of ulcerative enteritis.

A modified complement fixation test using tissue extract as antigen for the detection of infectious enteritis in quail is described by Morris (1948). The results of experimental trials and of tests of birds in a spontaneous outbreak of ulcerative enteritis in a flock of 403 quail gave results which indicate that the test may be used in the detection of carriers of the disease.

Dissemination—The disease spreads rapidly among susceptible birds, rendering it difficult in some cases to rear flocks. Bass (1939) has called attention to the prevalence of coprophagy among penned birds and this habit would tend to spread the condition rapidly. He was able to transmit the malady by feeding (1) droppings from diseased birds, (2) macerated diseased intestine, (3) remains of a yolk-sac from a nineteen-day-old affected quail, (4) pure cultures of the organism, and (5) by placing healthy birds in cages in which diseased quail had been kept. This author, although he was unable to isolate the bacillus from eggs, has observed ulcerative enteritis in very young birds, and believes there is some evidence that recovered birds become carriers and transmit the disease through the egg.

Treatment and Control—No treatment is known to be effective against ulcerative enteritis. Various sulfonamides including sulfaguanidine, sulfathiazidine and sulfasuxidine at 2 per cent concentration in the mash were shown by Morris

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Chapter 7

VIRUS DISEASES

AMONG the most troublesome diseases to which poultry are susceptible are those caused by filterable viruses. Some of these filterable agents are extremely virulent and the diseases induced by them are capable of causing severe losses to flock owners. Fowl pox, laryngotracheitis, fowl paralysis, infectious bronchitis, Newcastle disease, and fowl pest are among the most important conditions in this group.

FWL POX

Fowl pox (also known as chicken pox, contagious epithelioma, canker, avian diphtheria and sore-head) is a highly infectious disease of fowls, capable of manifesting itself in two distinct forms or types. The skin or cutaneous type is characterized by the presence of wart-like eruptions on the unfeathered portions of the body, particularly the comb, wattles, and eyelids. The diphtheritic or throat type (avian diphtheria) manifests itself in the form of membranous patches in the mouth and throat. It is not uncommon to find both types of the disease present in the same bird.

Cause.—Fowl pox is caused by a filterable virus designated as *Borreliota arium*. For many years the skin and diphtheritic types of the disease were regarded as distinctly separate conditions, and various agents such as bacteria and protozoa were believed to be the causative factors. In 1902, however, Marx and Sticker demonstrated that the causative agent of fowl pox is filter-passing and their findings were soon confirmed by others. Further proof that the etiological factors of the two conditions are identical is furnished by the fact that the chickens which recover from avian diphtheria are immune to the cutaneous form of the disease and *vice versa*.

In the epithelial cells of fowl pox lesions there are present minute, rounded or oval "inclusion bodies," and Woodruff and Goodpasture (1929) have demonstrated that a single

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Symptoms.—The symptoms produced by the disease depend upon which form of the condition is manifested. In the skin type, the first indication of infection is the appearance, principally on the comb or wattles of small grayish, raised, blister-like spots. These soon dry up into brownish scabs which gradually enlarge and eventually coalesce with adjacent lesions to form a larger wart-like eruption. If the scabs are removed, the surface beneath them is seen to be rough, raw and bleeding. These lesions may be few in number



FIG. 39 — Appearance of fowl pox lesions on the comb and wattles about ten days after natural infection.

or so numerous as to cover practically the entire unfeathered skin of the head including the comb, wattles, face, eyelids, earlobes and corners of the mouth. Similar eruptions may also appear on the legs and feet, about the vent, and under the wing. The scabs covering the lesions persist for some time, often for as long as three or four weeks, after which they gradually become detached, leaving a grayish, healing area. Fowls affected with this type of the disease may show little or no systemic disturbance if the infection is light, but if severe it is evidenced by listlessness, inappetence, loss of weight and marked reduction or complete cessation of production in laying hens.

one of these bodies is capable of inducing a typical fowl pox lesion when introduced into a feather follicle of a susceptible fowl. These workers have also shown that each of these inclusion bodies in turn contains hundreds of extremely small, definite particles (Borrel bodies) which they believe to represent the actual virus of the disease.

The virus of fowl pox is extremely resistant to drying and it is this property which enables it to remain virulent for a considerable length of time when exposed to external influences. The ability to resist desiccation is an important factor in the persistence of the virus on premises where the disease has occurred.

In cross-immunity tests Beaudette and Hudson (1941) used canary, pigeon, fowl, and turkey pox viruses. The tests were made upon six-weeks old chickens which were undersized because of inadequate diet and which were housed in quarters not altogether satisfactory. As a result of their work they concluded, "That canary virus immunizes against itself and against pigeon virus to a high degree. The canary virus seems to produce no immunity against turkey virus, but apparently a slight immunity to fowl virus. Similarly pigeon virus protects against itself and canary virus but does not give complete protection against turkey and fowl virus. Turkey and fowl viruses gave almost complete protection against the four viruses used."

Although closely related, the various pox viruses apparently differ in their biological properties. Pigeon virus is readily transmitted to fowls by artificial means, but it is more difficult to produce the disease in pigeons by the use of fowl virus.

Occurrence—Fowl pox is prevalent wherever poultry is raised. The disease sometimes assumes an exceptionally virulent form, affecting large numbers of fowls and causing serious losses. It is most prevalent during the fall and winter months, but may occur at any season of the year.

Susceptibility—Domestic chickens are highly susceptible to fowl pox, but the disease also affects turkeys, and to a less extent ducks, geese, pheasants, quail, canaries and hawks. Mammals are not susceptible to natural infection with avian pox virus.

surface. As the disease progresses the patches become larger, thicker and of a cheesy consistency, and may become so extensive as to prevent the fowl from closing its mouth. Interference with breathing, accompanied by rattling noises may be caused by the presence of patches on or near the larynx. If eating is interfered with to any extent, the affected fowls soon lose weight and present a picture of general unthriftiness.

Involvement of the eyes is not uncommon and is characterized at first by inflammation of the membranes and the presence of a watery discharge. This stage is often followed by the formation and accumulation of cheesy, yellow pus, causing the eye to bulge and giving it a puffy appearance.

Postmortem Appearance — Aside from the typical lesions of the skin and mouth, no constant changes are induced by pox infection in the organs of the fowl. It is quite probable that the presence of any changes other than the characteristic eruptions may be attributed to the action of microorganisms which gain entrance through the eroded skin or mucous membrane.

Diagnosis — The presence of typical wart-like nodules on the comb, wattles and other unfeathered areas, and of yellowish, adherent, membranous or cheesy patches in the mouth and throat usually enables a diagnosis to be made.

Transmission — In some outbreaks of the disease, dissemination occurs rapidly, with the majority of the fowls in the flock becoming infected within a short time, while in other instances the spread is slow. Close contact of birds aids in *rapid spread*, and it appears that the virus requires a break in the skin or mucous membrane for its entrance. Such abrasions are readily produced by injuries incurred during fighting or by the ingestion of sharp pebbles or particles of food.

It has been demonstrated that certain kinds of mosquitoes are capable of transmitting the virus of fowl pox. Kligler, Muckenfuss, and Rivers (1928) were able to induce the disease in susceptible birds rather regularly by allowing mosquitoes to feed on pox lesions and then causing them to finish their meal on healthy birds. Kligler and Aschner (1929) showed that mosquitoes which had fed on infectious material were capable of transmitting the virus for as long as sixteen

Johnson (1938) observed an unusual outbreak of fowl pox in six weeks old chicks in which practically all cases of the disease were characterized by the development of sores and scabs on the feet and legs. Very few of the birds had lesions on the skin of the head and in none were there any lesions in the throat. The author attributes the scarcity of pox scabs about the head to the lack of development of combs and



FIG. 40.—The diphtheritic type of fowl pox is characterized by the presence of firmly adherent patches in the mouth and throat. (Courtesy of Illinois Agricultural Experiment Station.)

wattles in the young chickens and offers the interesting thought that the failure of throat lesions to develop in a single fowl might possibly be due to some difference in strains of fowl pox virus as regards affinity for certain tissues.

The diphtheritic form of the disease is characterized by the formation of yellowish raised, membranous patches in the mouth and throat. These necrotic pieces of tissue are firmly adherent and, if forcibly removed, leave a raw, bleeding

membrane of a developing chick embryo. The fact that the virus can be produced free from bacterial contamination is important in avoiding some of the inflammatory reactions which sometimes resulted from use of the older scab virus.

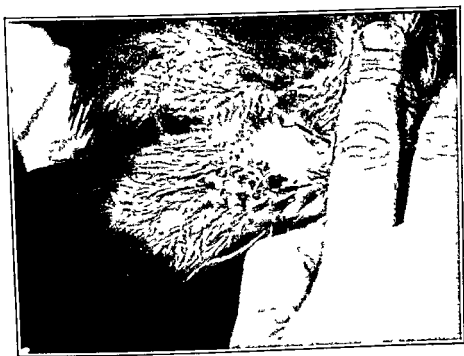


FIG. 41 —A "take" following successful vaccination with fowl pox virus is evidenced by the development of a brownish scab in from seven to ten days (Courtesy of Illinois Agricultural Experiment Station)

Brandly (1936 and 1937) has given interesting reports of his studies of egg-propagation of viruses. He found that continued passage of fowl pox virus by this means for as many as twenty passages did not alter the virus insofar as the appearance of the lesions induced by it in susceptible fowls was concerned. The "takes" and immunity induced by egg-propagated virus were just as satisfactory as were those engendered by scab virus.

Both fowl pox vaccine and pigeon pox vaccine are now produced by the egg-propagation method, and both are widely used in the control of fowl pox. Both are live-virus vaccines and must be used with care in order to avoid spread

days even though some of the insects had fed on animals of another species in the meantime. In a later study, the same authors (1930) were able to demonstrate the presence of fowl pox virus in mosquitoes caught near a house in which pox infected birds were kept. Matheson, Brunett and Brody (1932) also found that mosquitoes were capable of inducing fowl pox in susceptible birds for as long as twenty seven days after the insects had fed on comb lesions. There appears to be little doubt but that mosquitoes serve as vectors of the disease and as such may account for some of the outbreaks of the infection in which the method of introduction is obscure.

Mortality —The death rate from fowl pox varies from one outbreak to another. In some flocks the disease is severe and the losses are correspondingly large, while in others the majority of cases may be mild with few or no deaths. The greatest damage undoubtedly is caused in pullets about to lay or in hens already laying. In the former, production is greatly delayed while in the latter it may be curtailed or even stopped for a considerable length of time. The appearance of fowl pox in birds already suffering from some other disease or from parasitism always leads to more serious consequences than when the infection is uncomplicated.

Treatment —Medicinal treatment of fowl pox is of little avail although removal of lesions which interfere with eating and drinking may be helpful. The raw areas resulting from this treatment should be painted with tincture of iodine or with mercurochrome to facilitate healing. Manwell and Goldstein (1939) reported that they have successfully treated pox in canaries by the application of mercurochrome.

Prevention and Control —Fowls which have survived an attack of pox are in practically all cases rendered solidly immune for a considerable period of time thereafter. This fact served as the impetus in many attempts to perfect a vaccine against the disease and from time to time various preparations have been used for this purpose.

Early vaccines were made from dried scabs obtained from comb lesions but Woodruff and Goodpasture (1931) showed that a pure virus could be obtained by inoculating small amounts of germ free fowl pox virus into the chorioallantoic

Because of the fact that turkeys vaccinated against pox while still young birds frequently do not develop immunity sufficiently strong to carry them through the breeding season, Hinshaw (1941) recommends that those birds which are to be kept as breeding stock be revaccinated. The time at which to revaccinate depends upon various factors. In case the poults are brooded at all seasons of the year and the initial vaccination is done when the birds are eight to ten weeks of age, the second vaccination may be done when they are six to seven months of age. In the event most of the poults saved for breeders are hatched from March to June and first vaccinated in July or August, the second vaccination may be done after disposal of the market birds and just before the breeding pens are made up.

INFECTIOUS LARYNGOTRACHEITIS

Infectious laryngotracheitis (also known as infectious tracheitis, "flu") is an acute, highly contagious disease of fowls characterized by respiratory distress, rapid spread and, in many cases, high mortality. It is one of the few diseases of fowls against which a successful method of vaccination has been developed. The infection is one of the most serious maladies confronting the poultry industry today.

Cause.—The disease is induced by a filterable virus termed *Tarpeia avium*. Beaudette (1930) reported the cause of "infectious bronchitis" to be a filterable agent and from his description of the disease it is evident that the condition he was studying was infectious laryngotracheitis. Beach (1931) conclusively established the filterable nature of the causative agent of the disease. The virus is found in abundance in the exudates of the respiratory tracts of affected birds. Schalm and Beach (1935) have made rather extensive studies regarding the effects of various chemical and physical agents upon the virus of infectious laryngotracheitis. In one trial the virus in tracheal exudate showed no decrease in virulence after six hours exposure to direct sunlight, while in a second trial the virulence was lost in seven hours. Virus in tracheal exudate was inactivated by exposure for one minute to a 5 per cent solution of phenol; in thirty seconds by exposure to a 3 per

of the disease to unvaccinated birds. Pigeon pox vaccine produces only a mild reaction in fowls and may therefore be used on flocks of laying hens to produce a temporary immunity of two to three months without interfering with egg production. It is not effective for the immunization of turkeys against fowl pox.

The two methods of vaccination in common use have been well described by Hall (1956) as follows:

"In the stick method, two needles (about one-fourth inch apart in a cork or other holder) are dipped into the vaccine container and then applied by puncturing the wing web. This gives four cutaneous inoculations simultaneously, two on each side of the wing web since the needles pierce both layers of the skin. This method is fast, accurate, and economical of vaccine.

'In the older follicle method four or five feathers are plucked (usually from the upper lateral surface of the thigh) and the vaccine is applied to the feather follicles with a stiff bristle brush. The vaccinated birds should be checked for 'takes' a week to ten days after vaccination. A "take" is indicated by swelling and redness of the vaccinated follicles or the stick wounds."

If fowl pox is prevalent in the vicinity, it is considered sound practice to vaccinate all young chickens at six to twelve weeks of age. If the disease has not been a problem locally some flock owners prefer not to vaccinate routinely but to rely instead on pigeon pox vaccine if an outbreak of the disease occurs in the laying flock.

Natural outbreaks of fowl pox in commercial broiler flocks led Seeger and Price (1956) to test both pigeon pox and fowl pox vaccine on young chicks at one, five, ten and fifteen days of age. They found fowl pox virus entirely satisfactory when applied by the wing web method.

Pigeon pox vaccine does not appear to produce a solid immunity against subsequent pox infection in turkeys. Brandly and Dunlap (1938) found that although turkeys could be infected with pigeon virus, there was very little immunity against subsequent exposure to fowl virus. Hinshaw (1941) has also advised that fowl vaccine rather than that of pigeon origin be used when vaccinating turkeys.

sneezing, during which the fowl shakes its head vigorously in an attempt to dislodge the offending exudate which has accumulated in the larynx and trachea. The infected bird often assumes a sitting posture with the eyes closed, and during breathing a gurgling or rattling sound may be heard. At inspiration the head and neck are raised and extended and the mouth is opened, in an effort to take in as much air as possible. The inspiration of air is frequently accompanied by a loud, wheezing or whistling noise, leading to the designation of such birds as "callers." Upon expiration the head is lowered, often to the extent that the beak rests upon the ground. During the paroxysms of coughing and sneezing, masses of blood-tinged mucus or of clotted blood may be expelled, with temporary relief from the breathing distress. Excessive accumulation of exudate in the larynx and trachea may result in sudden death of the fowl from asphyxiation. In affected baby chicks respiration is exceedingly rapid and the mouth is opened widely at each inspiration.

The course of the disease usually ranges from seven to fifteen days, although symptoms may be manifested for as long as one month. Those fowls which recover from the infection gradually return to apparently normal health, but many such birds have been found to continue to harbor the virus in their respiratory tracts and thus become "carriers." Gibbs (1932) found carrier birds to be eliminating virus for as long as four hundred and sixty-seven days, during which time it was readily transmissible to susceptible chickens. In a later report the same author (1936) states that fowls may remain carriers for as long as two years. He found that the virus appeared to be confined to some portion of the larynx or trachea, and that while some of the carrier birds "rattled" when breathing, this symptom was not a reliable means of detecting chronic cases.

Postmortem Appearance.—The changes observed at autopsy are confined to the respiratory tract. There is often present about the nostrils a mucoid exudate, which may be dried into flakes. A sticky mucus is present in the throat and the larynx is inflamed, swollen and edematous. Upon opening the trachea, varying amounts of blood and blood-streaked exudate are found adhering to its wall. Pin-point hemor-

cent compound cresol solution and in the same time to a 1 per cent solution of sodium hydroxide.

Berch (1932) successfully cultivated the virus of infectious laryngotracheitis in a medium of minced chicken embryo in Tyrode's solution. Burnett (1934), Brandly (1935 and 1936) and others have reported in detail regarding cultivation of the virus upon the chorio-allantoic membrane of the developing chick. In this connection it is interesting to note that Brandly (1936) in inoculation trials found that the developing eggs of only chickens and turkeys were susceptible to laryngotracheitis infection while negative results were obtained with the eggs of pigeons, guinea fowls and ducks.

Occurrence. Laryngotracheitis is a comparatively new disease of fowls in the United States but it is already widespread and has been reported from many sections of the country. The disease also occurs in England, Canada, Germany, Australia and Hawaii. Severe outbreaks have occurred in many broiler plants and commercial feeding stations as well as in individual flocks. The condition was apparently first described as a disease entity by May and Littler (1926) who definitely differentiated it from fowl pest. Earlier reports of bronchitis are found in the literature but if the conditions described were in reality laryngotracheitis it evidently had not attained the severe form or wide distribution which characterize it today. Outbreaks are noted more frequently in the fall and the greater losses occur in the winter months in hens in production.

Susceptibility.—Chickens are extremely susceptible to laryngotracheitis. Hudson and Beaudette (1932) found pheasants and a pheasant bantam cross to be susceptible to the virus and it appears that these fowls with chickens are the only species definitely known to be susceptible. Seddon and Hart (1936) were unable to reproduce the disease in turkeys, ducks, starlings, quail, pigeons or sparrows but did induce infection in pheasants. All breeds of fowls appear to contract the disease readily and birds of all ages are susceptible although the incidence is greater in old than in young stock.

Symptoms.—The first noticeable symptoms of the disease are watery eyes and a tendency for the affected bird to remain quiet. This is soon followed by fits of coughing and

rhages may also be seen in the lining membrane of the inflamed larynx and trachea. If the course of the disease has been somewhat subacute, there is often present adhering to the rim of the larynx, a yellowish, cheesy exudate which may practically fill the laryngeal opening. Flakes of yellowish debris may also be found in the mouth or mixed with the tracheal exudate. Congestion of the lungs occurs in some cases but this change is not constantly present.

Diagnosis.—The diagnosis of laryngotracheitis is based upon the rapid spread of the condition in the flock and the presence of typical symptoms and lesions. The disease might readily be confused with other respiratory disturbances particularly "infectious bronchitis" of young chicks, or with the diphtheritic form of fowl pox, but the less rapid spread of the latter aids in the differentiation.

Transmission.—In many cases the means by which laryngotracheitis infection gains admission to a flock is not evident. Apparently healthy carrier fowls, if added to the flock, serve as a ready means of introducing the virus and such fowls serve to perpetuate the infection in the same flock year after year. It is also probable that the virus is transmitted on the clothing of persons who have visited premises on which the disease exists. Beaudette (1937) has also called attention to the possible rôle of contaminated feed sacks, crates, and other equipment in introducing the infection. Wild flying birds, going from one flock to another, must also be considered as potential mechanical spreaders.

There is no evidence that the virus is transmitted through the egg and Brandly (1934) has shown that apparently there is little danger of transmission by this means. Brandly and Bushnell (1934) were unable to demonstrate virus on the surface of eggs laid by a flock during an active outbreak of the disease, or on eggs from a flock harboring carriers. It appears from these findings that dissemination of the virus on the surface of eggs is not a factor in the spread of the infection. Brandly (1934) found that chicks hatched from eggs artificially inoculated at the tenth day of incubation were not affected and concludes that the danger of infection via the egg is not significant.



FIG 42 —Laryngotracheitis. Above characteristic position during inspiration. Below an advanced case in which the fowl is in a state of exhaustion. (Courtesy of California Agricultural Experiment Station.)

infection of the upper respiratory tract of birds held at 40° C in efforts to induce immunity, but with indifferent results. The same authors (1932) succeeded in infecting the lining of the cloaca and of the bursa of Fabricius by direct swabbing of the virus into the parts, and upon this fact the method of immunization so widely used at present is based. Molgard and Cavett (1947) have reported favorably on the vaccination of chickens by the feather follicle method. They found that the vaccine, when applied by this method, produces immunity just as it does when applied to the cloaca or to the Bursa of Fabricius. They state that the feather follicle method of vaccination gives a higher percentage of readable takes than is produced when the birds are vaccinated in the cloaca.

Fowls vaccinated by the cloacal method developed a typical cloacal inflammation ("take") which ran its course in about one week and was followed by rapid development of immunity. ("Takes" following vaccination vary widely in their manifestations, ranging from a simple, mild inflammation of the mucous membrane to a pronounced reaction characterized by the presence of a fibrinous pseudomembrane and severe inflammation of the lining membranes of the bursa or cloaca or both.) Birds so treated did not develop the respiratory form of the disease. The vaccine used in this work consisted of the dried tracheal exudate obtained from infected birds, suspended in 50 per cent glycerin for use. Vaccination was carried out by swabbing the vaccine into the cloaca with a rotary motion sufficient to induce slight abrasion of the lining membrane. The authors point out that there is no method for determining the activity of the vaccine except by actual trials on susceptible fowls. To insure successful results they also stress the necessity of obtaining 100 per cent of "takes" because of the danger of vaccinated birds transmitting the infection, with subsequent respiratory involvement, to those fowls which do not give a vaccination reaction.

The most desirable time for vaccination appeared to be when the birds were between two and three months of age. The inoculation of baby chicks by the cloacal method was not satisfactory because of the difficulty in obtaining "takes" and also because of the development of a peculiar, unex-

Mortality — Available figures indicate that mortality may range from less than 5 per cent to as high as 60 per cent. In a study of 25 outbreaks involving 14,574 chickens Hinshaw, Jones and Graybill (1931) found an average mortality of 13 per cent and there was also a considerable loss because of lowered egg production in the infected flocks.

Treatment — Numerous methods of treatment for larvngotracheitis have been tried usually with unsatisfactory results. Relief has been gained in some instances by raising both the temperature and humidity of the air in the house in which the birds are kept. The application of such agents as mercurochrome, argyrol, tincture of iodine and pine oil antiseptic to the inflamed throat has had no beneficial effect on the course of the disease and often leads to increased respiratory distress. It may therefore be stated that at present no method of treatment has been found to possess sufficient merit to warrant its recommendation.

It has been shown by Beach (1931) and Brandly (1934) that the serum of immune fowls possesses some value in neutralizing the virus and it is possible that the use of such serum if available might be indicated in the treatment of exposed flocks. So far as the writers are aware however this immune serum is not yet available commercially and it is quite probable that the cost of producing such a serum would prohibit its general use.

Vaccination — The discovery of the fact that the cause of larvngotracheitis is a filterable virus explained the failure of mixed bacterins as immunizing agents and directed the attention of investigators to the possibility of developing a virus vaccine. Kernohan (1930) injected subcutaneously various suspensions of tracheal exudate which had been attenuated by either heat or chemicals but stated that none of the preparations exerted any immunizing effect. Gibbs (1933) found that subcutaneous and intravenous injections of virus filtrate brought about immunity in some instances but the difficulty of administration and the uncertainty of results did not warrant their use in the field.

Beaudette and Hudson (1933) reported having tried the subcutaneous injection of active virus and of virus modified by passage through another host, infection of the eye and

ing small doses of virus intermittently into some part of the body other than the respiratory tract, but concluded that the inoculation of the bursa of Fabricius appeared to be the most satisfactory method for creating immunity to the disease.

Although it appears possible to produce a solid degree of immunity to laryngotracheitis by the cloacal or bursal method of vaccination, these methods are not without potential danger. The vaccine, as produced at present, is capable of inciting severe infection of the respiratory tract and for that reason should be used only by veterinarians who fully understand its indications and application. The indiscriminate use of the vaccine might readily lead to severe outbreaks of the disease and careful consideration of this fact should be given to all cases in which vaccination is contemplated. It is hoped that further studies will reveal a method for producing a vaccine which is safe and at the same time effective in combating one of the most troublesome of poultry diseases.

Prevention and Control.—Practical measures for the prevention and control of laryngotracheitis consist of strict application of the principles of hygiene and sanitation. Gibbs (1933) reports that poultrymen in Massachusetts have been very successful in controlling the disease by use of the "Massachusetts plan," which embodies the following measures:

1. All birds on the premises having had infectious laryngotracheitis or having been exposed to it should be condemned and disposed of.

2. Incubators and brooders should be cleaned, disinfected and isolated at some distance from the condemned birds.

3. Chicks not having been exposed to infectious laryngotracheitis may be kept for restocking, provided they are entirely separate from the condemned birds and the premises occupied by them.

4. All buildings occupied by the condemned birds should be thoroughly cleaned and disinfected as soon after vacating as possible.

5. The houses and yards, after being cleaned and disinfected, should be opened to the air and sunshine and left vacant for two months or longer.

plained swelling and blackening of the eyes. The vaccination of six-months-old fowls also failed to give a satisfactory number of reactions.

Because they were unable to induce "takes" in all susceptible fowls by cloacal inoculation, Beach, Schalm, and Lubbehusen (1934) tried introducing the vaccine directly into the bursa of Fabricius by means of a syringe fitted with a blunt-pointed, slightly curved, rather large hypodermic



FIG 43 —Showing the cloacal method of vaccinating fowls for the prevention of laryngotracheitis

needle. It is the opinion of these authors that this method possessed some advantages over cloacal vaccination in that it was readily accomplished; less virus was required; a rather uniform dosage could be employed; and the percentage of "takes" was very satisfactory. The reactions following this method of inoculation did not differ essentially from those induced by the cloacal method, and the resulting immunity appeared to be very solid.

In his studies of vaccination, Gibbs (1933) found that immunity to laryngotracheitis may be induced by introduc-

dividual chickens showing two or even three forms of the disease.

Occurrence.—The disease has been reported from practically every section of the United States, as well as from every other country in which poultry raising is carried on to any degree.

Susceptibility.—Chickens appear to be the only species of domestic birds naturally susceptible to the disease. Jungherr (1939) reported the presence of a condition resembling avian leukosis complex in a flock of ring-necked pheasants and Johnson (1941) was able to induce the neural type in one, and the visceral type in two pheasants of a group of twenty-six which he inoculated with affected nerve tissue.

It has been held that all breeds of chickens are equally susceptible to the disease, but Davis *et al.* (1947) in their studies of postmortem diagnoses of many thousands of birds found a higher degree of susceptibility to the visceral type in Barred Plymouth Rocks than in White Plymouth Rocks, White Leghorns, and Rhode Island Reds. They also noted that the average incidence of the neural and ocular types was highest in the White Leghorns, slightly less in Rhode Island Reds, and considerably less in Barred Plymouth Rocks.

It appears that the younger the bird, the greater is the susceptibility, with the greatest incidence being in fowls between four and ten months of age. Biely, Palmer, and Lerner (1933) observed the disease in chicks less than sixty days of age and one chick was found to be affected when only thirty-seven days old. Usually, however, avian leukosis complex is slow in developing and symptoms are not observed before the chickens are two to five months of age.

The disease apparently affects fowls of both sexes with about equal frequency in most outbreaks, although there have been reports which suggest that the incidence may be higher in females than in males in some instances. In this connection, Burmester and Nelson (1945) obtained interesting and possibly significant results by using sex hormones. As a result of their experiments, these workers noted that the male hormone used increased the resistance of males and capons to lymphomatosis and they state that this fact may

6 Overalls and shoes worn around the condemned birds or on the premises occupied by them should not be used in the houses, on the range, when handling feed, or when caring for chicks for restocking, unless laundered or thoroughly disinfected

7 Utensils and equipment used around the condemned birds, or in cleaning and disinfecting the premises occupied by them, should not be used in the houses or on the range where the chicks for restocking are kept, unless said utensils and equipment have been cleaned and disinfected thoroughly

8 United States Department of Agriculture permitted disinfectants should be used as directed for disinfecting all poultry houses, utensils and equipment

9 New stock should be introduced from absolutely clean flocks, or from the poultryman's own hatchings which have been adequately protected from infection

10 The subsequent reintroduction of infectious laryngotracheitis should be carefully guarded against

THE AVIAN LEUKOSIS COMPLEX

The term avian leukosis complex is used to designate a widespread, transmissible disease-complex of chickens. The malady is the cause of enormous financial loss to the poultry industry and by some is considered to exact a toll equal to or surpassing that of all other poultry diseases combined. The condition is characterized by various manifestations, including paralysis of the legs and wings, the formation of tumorous masses in different parts of the body, involvement of the eye with partial or total blindness, alterations in some of the bones, and in some cases by marked changes in the cellular structure of the blood. By some investigators avian leukosis complex and these blood alterations are held to be but manifestations of the same etiological agent, while by others they are considered as distinct but closely allied conditions.

Cause—The cause of the avian leukosis complex is a filter-passing agent or agents, submicroscopic, and believed to be viruses. Two or three forms of lymphomatosis are often present in a single flock, and it is not unusual to find in-

Durant and McDougale (1947) describe "soiled fronts" as a symptom of the neural or fowl paralysis type of the disease. The feathers under the mandible, along the throat, and in the region of the crop appear damp, darkened and permanently discolored, with this symptom apparently having been caused by involvement of the nerves controlling the salivary or other glands of the mouth. This nerve involvement permits escape of fluid through the corners of the mouth and a resultant flow down the front of the bird, and thus causes soilage of the feathers. The same authors also list wilted combs as a symptom of the neural type of the condition.

If the brain is involved, there may be manifestations of excitability or of sleepiness, and the head is moved from side to side or back over the body. Involvement of the neck muscles is evidenced by twitching of the head or inability to raise the beak from the ground. Affection of the nerves controlling respiration may cause difficult breathing, while digestive disturbances, as evidenced by flaccid crop and diarrhea are not unusual.

Ocular Type.—Involvement of the eye is common and is characterized by a change in the color of the iris from that characteristic of the breed to varying shades of gray, the condition then being referred to as "white eye" by poultrymen. In severe cases the pupil fails to respond to light and remains fixed, usually contracted but occasionally dilated, as compared with its normal accommodation to varying light intensities. The affected eye may bulge out and impairment of vision even to total blindness follows. In many cases the eye changes are the first indication of the disease, and in some instances constitute the only visible symptom. It is well to keep in mind that the irises of fowls are usually gray during the first few months of life, with pigmentation or coloring appearing later, therefore lack of this coloring in a young bird does not necessarily indicate the presence of the ocular type of the avian leukosis complex.

Visceral Type.—All internal organs of the fowl are susceptible to involvement by lymphomatosis. Characteristic of this type of involvement is the presence of tumorous masses which range in size from very small, even microscopic,

account, in part at least, for the lower incidence of the disease among males than among females

Types of the Disease—The extremely complex nature of the disease has resulted in much confusion regarding the terms used to designate the various manifestations of the condition. The following outline was adopted by investigators at a conference held in 1940 at the United States Regional Poultry Research Laboratory in East Lansing, Mich., and later (1941) revised. The outline refers to the pathological manifestations of the disease and represents an attempt to standardize the nomenclature used in describing these manifestations

Term	Synonym
1 Lymphomatosis	Lympholeukosis
a Neural	Fowl paralysis
b Ocular	White eye
c Visceral	Big Liver Disease
d Osteopetrotic	Marble bone
2 Erythroblastosis	Erythroleukosis
3 Granuloblastosis	Myeloleukosis
4 Myelocytomatosis	

Symptoms—The manifestations of the lymphomatous type of the avian leukosis complex are readily observed clinically or upon postmortem examination, while the other types usually require laboratory determination

Neural Type—The symptoms observed in fowl paralysis depend upon the part of the body affected. Lameness in one or both legs or slight drooping of a wing are usually the first indications of the neural type. As the disease progresses, the lameness becomes more marked and the affected fowl, no longer able to stand, lies on the ground. The legs are often held in peculiar positions and may be extended forward or backward or out from the body. Limpness of the legs and wings is commonly noted and the fowl seems to have little or no control over the parts. Shrinking of the muscles of the involved appendage is sometimes severe, leaving the part withered and atrophied. A peculiar jerking and twitching of the affected limbs may also occasionally occur.

affected. Some of the changes are of such a nature as to require microscopic examination of nerves, blood, bone, and tumor-like masses.

Neural Type — The large nerves, as well as the plexuses of the wings and legs, especially in those cases showing paralytic symptoms, often show definite alterations. They are swollen, even to several times their normal size, appear water-soaked, and are changed in color from milk-white to a yellowish or gray color. The cross striations characteristic of normal nerves are lost, and nodules of varying size may be found at any point along the affected nerve. In the large sciatic nerve of the leg it is not unusual to find one of the divisions markedly enlarged and edematous, with no apparent changes occurring in the division next to it. These alterations are caused by the infiltration and deposition of large numbers of lymphoid blood cells which are produced in excess and subsequently deposited in various organs of the body. In contrast to the gross alterations which are readily seen upon examination, the involvement of nerves in some cases is of such a nature as to necessitate microscopic examination for detection.

Ocular Type — When ocular involvement is present there is a definite lack of pigment in the iris. Its color is gray, and a deposition of lymphocytes is responsible for the color change. The eye may bulge out from its socket and one or both organs may be so affected.

Visceral Type — Tumorous masses of tissue are present in this type of the disease and may involve any of the organs or tissues such as the ovary, liver, spleen, kidneys, testes, lungs, mesentery, intestines, gizzard, pancreas, muscles, skin, lungs, and heart. These tumors are grayish-pink or yellowish in color and those visible vary in size from that of a very small nodule to a mass as large as a tennis ball. They may be so extensive as practically to obliterate an organ and this is particularly true when the ovary is affected. Some of the masses are fairly firm in consistency while others are soft and flabby. Upon incision, the center of such a mass is frequently found to be necrotic and yellowish in color. In severe cases it is not uncommon to find the abdominal

nodules to large tissue masses which occupy the greater part of the body cavities. The symptoms induced by the presence of these tumor like masses are not characteristic. Diarrhea is frequently present. It is not uncommon to find in an affected fowl extensive lymphomatous involvement of organs without any discernible symptoms having been displayed by the living bird.

Bone Type — A cautious jerky gait is usually the first symptom of involvement of the bones by the avian leukosis complex. The long bones of the legs and wings are the ones affected and in advanced cases the changes are readily apparent. The bone or bones are enlarged misshapen and puffy particularly those of the legs. Jungherr (1935, 1938) was apparently the first to associate this osteopetrotic condition with the avian leukosis complex.

Blood Types — As has been stated the various blood types of the avian leukosis complex can be determined with positiveness only by laboratory examination since none of them induces symptoms which can be considered diagnostic.

Erythroblastosis is usually manifested by paleness or yellowish discoloration of the comb and wattles and in most instances a rapid loss of weight. Despite the fact that the appetite usually remains good progressive weakness and emaciation occur until the bird is no longer able to stand. Diarrhea is as a rule present and the affected fowls are sleepy and the eyes are kept closed. Such cases may linger in this condition for some time or if the progress of the disease is rapid death may ensue within a few days after the initial symptoms are noted.

Granuloblastosis may be manifested by symptoms similar to those noted above for erythroblastosis and is usually seen in birds above six months of age. Yellowish discoloration of the comb and other unfeathered parts of the head is a rather common manifestation.

Myelocytomatosis designates an aleukemic tumor like form of the disease and does not give rise to any symptoms which are at all diagnostic or characteristic.

Postmortem Appearance — The changes observed at autopsy in cases of avian leukosis complex will as in the case of symptoms depend upon what part or parts of the body are

turgid or a soft, flabby consistency. This is the condition frequently referred to by poultrymen as "big liver disease." Small brownish or gray areas are found over the surface and in the substance of the organ. The capsule is easily torn and not infrequently death is found to have been caused by internal hemorrhage from the ruptured part. The spleen may also be tremendously enlarged, being flabby and pulpy in

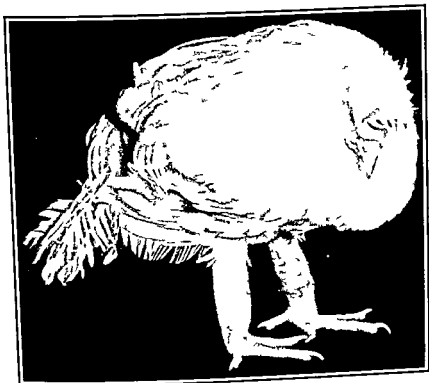


FIG 45 —Marble bone—such enlargement of the shanks and other bones in fowls is caused by osteopetrotic lymphomatosis (Brandly, Waters and Hall, *United States Dept Agriculture Yearbook*, 1942)

consistency. When cut, the substance of the organ is shiny and bulging and the color varies from a reddish-brown to a dark purple. Not infrequently the kidneys are affected, being swollen and having grayish or yellowish areas present in their tissue. The mucous membrane of the intestine, particularly the duodenal portion, may be studded with small hemorrhages and if the walls of the bowel are involved, they are thickened, grayish and easily torn.

organs so involved that it is practically impossible to separate the different structures.

Bone Type.—The long bones of the legs and wings are those usually altered in the osteopetrotic type of the disease. They are greatly thickened and enlarged but the length is not increased. The walls are much harder than normal and the marrow space is greatly reduced in capacity. The structure of the bony tissue is spongy in appearance but not in consistency.



FIG. 44.—Lymphoid tumors in the liver and intestines of a chicken a result of visceral lymphomatosis (Brandly, Waters and Hall, *United States Dept. Agriculture Yearbook*, 1942)

Blood Type.—The postmortem changes observed in the leukotic type of the malady are found principally in blood, liver, spleen, bone marrow and kidneys. The blood is thin and watery in appearance and microscopic examination of it reveals marked changes in the numbers of the various constituent cells, as well as the presence of cells which are definitely pathologic in character.

In the blood type of the disease, the liver is often enlarged to an enormous size, is grayish-red in color, and has either a

Alterations in the bone marrow are evidenced by a change in color from the usual blood-red to brick-red.

Diagnosis.—The occurrence of the symptoms and lesions described above is suggestive of the presence of some form of the avian leukosis complex but laboratory examination is necessary for positive diagnosis.

Transmission.—Visceral lymphomatosis is transmitted both by simple contact and through the hatching egg. The virus is shed in the feces and saliva of infected chickens, as shown by work at the Regional Poultry Research Laboratory. It was possible to induce a high incidence of visceral tumors by applying the inoculum to the nasal passages, conjunctiva, mouth, trachea and cloaca, and by spraying the virus into the air breathed by baby chicks.

Neural lymphomatosis is transmitted chiefly by contact, and young chickens are especially susceptible. Incidence of this form of the disease can be greatly reduced by rearing chicks in isolation from infected stock or premises.

Little is known concerning transmission of ocular lymphomatosis.

Mortality.—Recovery from the avian leukosis complex rarely occurs although a few instances are mentioned in the literature in which affected birds apparently survived. The losses are correlated with the number of fowls involved. In some outbreaks only a few birds contract the disease while in others a large percentage of the flock is involved. Losses frequently are so great as to render it inadvisable to continue with the flock at hand, and as has been stated, the disease probably results in more financial loss to the poultry industry than all other diseases combined.

Treatment.—At the present time no treatment is known to have any beneficial effects in combating the condition. There is evidence that recovered fowls have in their blood stream substances capable of neutralizing the causative agent. Although ducks and turkeys are not naturally susceptible, it has been shown by Lee (1941) that the injection of these species with leukotic material results in the production of substances in the cell-free plasma of their blood which are capable of inactivating the fowl leukosis agent. Olson (1940) states that the serum of recovered chickens and also that of

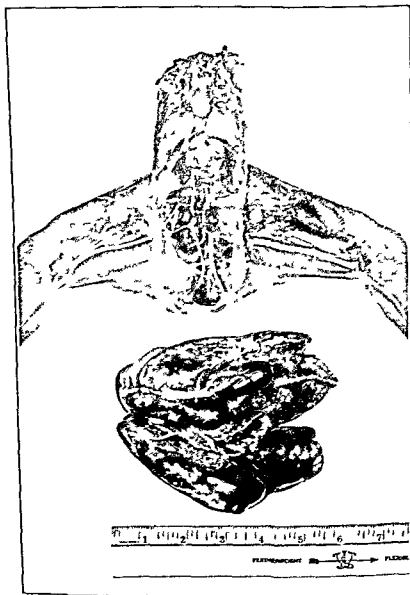


FIG. 46.—A severe case of avian leukosis complex showing tumors in the ovary and kidney. The enlarged nerve trunks at the right may be compared with the normal ones on the left. Below, the visceral surface of a greatly enlarged liver. The enormously enlarged gall bladder is not necessarily associated with fowl paralysis, but the spleen in affected fowls is often much larger than the one shown here.

first two weeks of life, has been stressed by Hutt *et al* (1944). The incidence in chicks reared in an environment over 100 feet from adult fowls was noticeably lower than in chicks kept closer to the mature stock. The evidence offered by these experiments, as well as that obtained by other workers offers a sound basis for following a program of keeping young chicks severely isolated from older birds.

3 The fact has been well-established that there are strains of birds which are resistant to the disease and that this characteristic can be transmitted to their offspring. The importance of this phase of control has been demonstrated and emphasized by Taylor *et al* (1943), Waters (1945) (1947), Waters and Prickett (1946) and Hutt, Cole, and Bruckner (1945). The practical application of selection of breeding stock which is resistant to avian leukosis complex should be an integral part of any control program.

NEWCASTLE DISEASE

Newcastle disease was first recognized as a distinct and separate disease of chickens in 1926 after outbreaks occurred in England and in the Dutch East Indies. It was recognized in various parts of the world and in 1940 a condition which closely resembled that disease was observed in flocks in California. At that time it was designated as "respiratory-nervous disorder" by Berch (1942) and Stover (1942) but the designation was soon changed to avian pneumoencephalitis. In 1944 it was shown that the virus of pneumoencephalitis was identical to the virus of Newcastle disease. In 1945 the disease was found on the Eastern seaboard, in New Jersey and New York. By 1947 the infection had been recognized in all sections of the United States.

Cause — The etiologic agent of the disease has been definitely established as a filterable virus designated as *Tortor furens*. It is readily cultivated in developing chick embryos and does not appear to be very resistant to natural conditions. Tilley and Anderson (1947) tested various chemicals for their power to destroy the virus. They found that sodium hydroxide 2 per cent, sodium orthophenylphenate 1 per cent, liquor cresolis saponatus 1 per cent, and quaternary ammo-

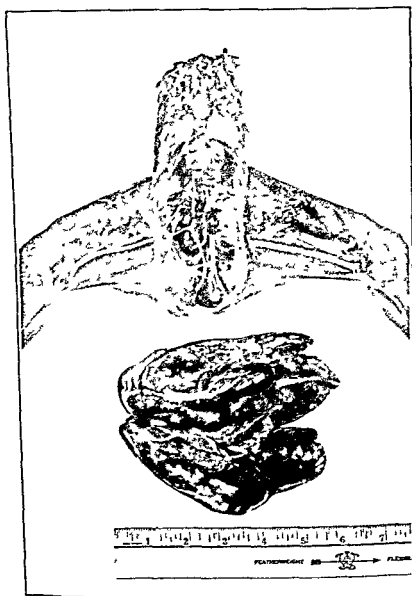


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ducks which have been injected with infectious material, are capable of neutralizing the etiologic agent *in vitro*

Burmester (1947) was able to demonstrate a definitely cytotoxic activity against lymphoid tumor cells of anti-serum obtained from birds which had been injected with lymphoid tumor antigen and Burmester, Prickett and Belding (1946) demonstrated that plasma from birds hyper-immunized against a lymphoid tumor, when injected into chicks that had received implants of the same tumor strain would reduce the mortality resulting from tumor involvement

Despite these findings there does not appear to be much hope for the control of avian leukosis in the field of serum therapy. Prohibitive costs of producing protective serum by the means at hand as well as the unknown degree and length of protection conferred by its injection offer little promise for control in that field

Prevention and Control.—A few attempts have been made toward controlling the avian leukosis complex by vaccination but to date the meager results have not been promising

Burmester and Waters (1956) state flatly that "None of the various forms of lymphomatosis can be prevented by vaccines available in 1956 or by the use of other prophylactic measures. No chemotherapeutics, antibiotics or other therapeutic treatments are known to be effective in the cure of chickens with this disease"

Sanitation, strict isolation of young birds and breeding for resistance appear to be the most promising measures for preventing and controlling the avian leukosis complex, although other factors must be considered in any attempt to control the disease

1 *Sanitation*—As is the case in dealing with any disease the importance of sanitation cannot be too strongly stressed. Convincing evidence that the causative agent of avian leukosis complex is often eliminated in fecal material emphasizes the importance of preventing contamination of feed, water, litter, and surroundings by this means

2 *Isolation of young birds*—In attempts to control the disease the importance of rearing young birds under strict isolation apart from mature fowls, particularly during the

first two weeks of life, has been stressed by Hutt *et al.* (1944). The incidence in chicks reared in an environment over 100 feet from adult fowls was noticeably lower than in chicks kept closer to the mature stock. The evidence offered by these experiments, as well as that obtained by other workers offers a sound basis for following a program of keeping young chicks severely isolated from older birds.

3. The fact has been well-established that there are strains of birds which are resistant to the disease and that this characteristic can be transmitted to their offspring. The importance of this phase of control has been demonstrated and emphasized by Taylor *et al.* (1943), Waters (1945) (1947), Waters and Prickett (1946) and Hutt, Cole, and Bruckner (1945). The practical application of selection of breeding stock which is resistant to avian leukosis complex should be an integral part of any control program.

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3. The fact has been well-established that there are strains of birds which are resistant to the disease and that this characteristic can be transmitted to their offspring. The importance of this phase of control has been demonstrated and emphasized by Taylor *et al.* (1943), Waters (1945) (1947), Waters and Prickett (1946) and Hutt, Cole, and Bruckner (1945). The practical application of selection of breeding stock which is resistant to avian leukosis complex should be an integral part of any control program.

NEWCASTLE DISEASE

Newcastle disease was first recognized as a distinct and separate disease of chickens in 1926 after outbreaks occurred in England and in the Dutch East Indies. It was recognized in various parts of the world and in 1940 a condition which closely resembled that disease was observed in flocks in California. At that time it was designated as "respiratory-nervous disorder" by Beach (1942) and Stover (1942) but the designation was soon changed to avian pneumoencephalitis. In 1944 it was shown that the virus of pneumoencephalitis was identical to the virus of Newcastle disease. In 1945 the disease was found on the Eastern seaboard, in New Jersey and New York. By 1947 the infection had been recognized in all sections of the United States.

Cause.—The etiologic agent of the disease has been definitely established as a filterable virus designated as *Tortor furens*. It is readily cultivated in developing chick embryos and does not appear to be very resistant to natural conditions. Tilley and Anderson (1947) tested various chemicals for their power to destroy the virus. They found that sodium hydroxide 2 per cent, sodium orthophenylphenate 1 per cent, liquor cresolis saponatus 1 per cent, and quaternary ammo-

num compounds Nos. 1 and 2 in 0.1 per cent solution were effective under laboratory conditions for killing the virus. The virus is found in the brain, spleen, lungs, tracheal exudate and air sac coverings of affected birds.

Occurrence—The disease occurs in many countries of the world and is very prevalent in the United States having been reported from practically every state.

Susceptibility—Chickens and turkeys are naturally affected as well as other species of birds: pheasants, ducks, geese, pigeons, quail, partridges, sparrows and other free flying birds. Birds of all ages are susceptible to the infection. The infection of human beings has been recognized only as a relatively mild pink-eye-like infection contracted by handlers of sick or slaughtered birds or by diagnosticians or laboratory personnel working with the virus.

Symptoms—The first symptoms usually observed in young birds are sneezing, gasping and often droopiness and it is in this stage of the disease that the manifestations rather closely resemble those of infectious bronchitis. Within a short time after the respiratory symptoms have appeared, nervous manifestations become apparent and are of three general sorts: (1) muscular incoordination, particularly of the neck and legs; (2) tremors or shaking of the entire body, and (3) total or partial paralysis of one or both legs. Affected fowls may display symptoms falling into any one or all of these categories. Some birds which are able to walk, eat and drink go through many peculiar motions such as walking backward or in circles with a constant twisting of the head and neck. The head may be drawn back over the body to the side or even under the body.

In older birds the first symptoms are usually coughing and a throat rattle followed by impairment of appetite and a rapid decline in egg production. Soft-shelled eggs and eggs in various stages of development may be seen and in some instances a partial moult has been observed. Lorenz and Newlon (1944) noted that some of the eggs laid by infected hens failed to form normal air cells, the air being present in free-floating bubbles. Other workers: Berg *et al.* (1947) Parnell (1950) found that eggs from hens that experienced an outbreak of Newcastle disease had a decrease in albumin

quality, and a production of abnormal shells tended to be permanent. The eggs did not keep as well in storage.

Recent studies indicate the virus may vary in virulence and may be present in a flock in such a mild form as to produce no apparent symptoms. The only way the infection is detected is by obtaining positive evidence in laboratory tests.

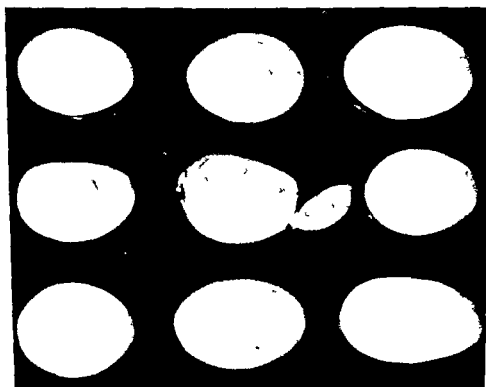


FIG. 47.—Misshapen or soft shell eggs are often produced by flocks infected with Newcastle disease or infectious bronchitis. (Courtesy of Minnesota Agricultural Experiment Station.)

Postmortem Appearance.—Clouding and thickening of the air sacs, mesentery, and other internal membranes are common findings at autopsy. These tissues may be yellowish in color, slimy, and covered with an adherent, frothy exudate. Yellowish or clear mucus may be present in the mouth, trachea or bronchi, and the lungs are often congested. Strings of mucus may hang from the mouth and in some cases there is a severe tracheitis. In very acute cases, small hemorrhages

may be present on the heart, intestines and other organs although this finding is not common. In mature fowls egg yolk may be present in the abdominal cavity either in cheesy chunks or in the fluid state.

Diagnosis—The occurrence of respiratory distress in fowls accompanied or followed by the nervous symptoms above described with a marked drop in egg production in laying hens points strongly to the presence of the disease. The respiratory symptoms might at the onset be readily mistaken for that of infectious bronchitis and possibly for laryngotracheitis while the nervous manifestation could be very similar to those seen in avian encephalomyelitis or in nutritional encephalomalacia.

Positive identification of the disease must depend on laboratory procedures since clinical symptoms are inconstant and cannot be considered pathognomonic. Four procedures are at present employed in the laboratory for the detection and identification of Newcastle disease as follows: (1) isolation of virus, (2) hemagglutination test (HA), (3) hemagglutination inhibition test (HI) and (4) serum neutralization test (SN). (See Chapter 5, p. 82, Diagnostic Methods.)

A test has been devised by Schmittle and Millen (1948) for the detection of hemagglutination inhibiting antibodies in unincubated eggs. They used egg yolk which had been treated with ethylene dichloride and ether as a substitute for the serum in the hemagglutination inhibition test and found that the previous presence of Newcastle disease in a flock could accurately be determined by the method. Among the advantages described by the authors are: no handling of birds, no blood required, does not require embryonated eggs and is less expensive and laborious.

Dissemination—It has been definitely established by De Lay, De Ome and Bankowski (1948) that the disease is air borne. They took samples of air from chicken houses in which evidence of Newcastle infection had been manifested seven to ten days earlier. They drew air samples through embryo fluids and treated the latter with antibiotics to kill the bacteria present. Subsequent injection of the material into chick embryos demonstrated the presence of the virus.

Affected fowls which are sneezing and coughing can thus readily communicate the disease to susceptible birds.

Evidence that the infection can be transmitted through the egg has also been obtained. Isolation of the virus from yolk-sacs of young chicks, dead embryos, and from infertile eggs laid by infected hens appears to offer plenty of supporting evidence for the theory that egg transmission does occur. On the other hand eggs laid by immune fowls have been found to possess specific antiviral activity, by Brandly, Moses, and Jungherr (1946). They observed that chicks from such eggs were relatively immune for two weeks after hatching, after which there was a rapid decline in resistance with only a small proportion of the chicks being refractory to infection after five to six weeks.

There are a number of additional ways the virus of Newcastle disease may be spread from an infected flock such as by contaminated poultry crates, equipment, feed sacks and free flying birds. Cats and dogs can act indirectly as carriers of the virus from one place to another. The flock owner himself may introduce it by carrying the virus home on his shoes and clothing. Chicks may be exposed during shipment by rail, truck, or air.

Treatment.—At present no medicinal treatment has been advanced as having any value. Proper housing and general good care are indicated in the effort to shorten the duration and severity of the infection.

Vaccination.—Numerous vaccines have been developed for the vaccination of poultry. A vaccination program is a valuable supplement to long range sanitation and management measures but must not be considered a replacement. There are two general types of Newcastle disease vaccines, the killed virus and the live virus (Van Roekel, 1955), (Fabricant, 1955), (Johnson, 1955).

The killed virus vaccines are prepared by growing suitable strains of virus in embryonating eggs, harvesting the tissues and killing the virus with chemicals or ultraviolet light. The killed vaccines have the advantage in that they will not spread the disease but have the limitation that the immunity will not last and may wane in two to six months after vaccination. Revaccination after two to three months is highly

desirable to obtain long immunity. Each bird must be handled and injected.

The living virus vaccines are prepared by growing modified strains of the Newcastle disease virus in embryonating eggs. There are several strains used in preparing the present commercial vaccines. The modified virus employed in the 'stick' method vaccine may produce mild to severe reaction in some birds. It has been generally replaced by the more modified strains that are applied to individual birds by intranasal, intraocular or intramuscular routes or by mass techniques of applying the vaccine by spray, dust or in the drinking water.

Prevention. Every precaution must be taken to avoid the introduction of Newcastle disease into a hatchery or onto the poultry farm or broiler plant.

FOWL PLAGUE

Fowl plague (fowl pest) is a highly acute infectious disease of fowls characterized by an extremely rapid course and high mortality. In some instances it resembles fowl cholera in its manifestations but it can be readily differentiated from the latter by appropriate laboratory examination.

Cause.—The disease is caused by a filterable virus which is highly infectious even in minute quantities, and which appears to be intimately associated with the red blood cells in diseased birds. The virus is contained in the blood, nasal exudate, liver, droppings and nervous system and thus may escape from the body by several channels. Microscopic amounts of the virus—even as little as $\frac{1}{1000000}$ cc. of unfiltered blood—have been found capable of inducing the infection when injected into a susceptible fowl. The resistance of the virus is not marked and disinfectants in the strengths ordinarily used apparently destroy it quite readily, especially if they are used while very hot. Fowl pest virus is not so resistant to drying as are some of the other viruses, although it does survive desiccation for some time. Purchase (1931) found that the infective agent survived for two hundred and eighty-seven days in flesh and for three hundred and three days in bone marrow when kept at a chilling temperature.

He also found that the virus survived for as long as eighteen days on feathers plucked from a recently dead bird and suggests that spread might be effected in this manner. Burnet and Terry (1934) were able to propagate the virus of fowl plague by inoculation of the chorio-allantoic membranes of developing chicks. As is the case with certain other viruses, this technique offers definite advantages for studying the filterable agents.

Occurrence — Fowl plague is prevalent in Europe, particularly in Germany, France, Belgium and Austria. A rather severe outbreak of the disease occurred in the United States in 1925 and was confined principally to some of the eastern states, although it also appeared in some areas in the mid-west. Another outbreak was reported in New Jersey in 1929. In both instances the disease was eradicated by prompt and vigorous control measures on the part of sanitary officials and has not been known to recur.

Susceptibility — Chickens, turkeys and geese are highly susceptible to fowl plague, but guinea fowls, pheasants, blackbirds, ducks, sparrows, owls, hawks and other birds also contract it. Wild waterfowl and pigeons are somewhat more resistant, although the disease has been produced in them by artificial inoculation and even pen contact in some cases. Mammals are not susceptible to the infection and man also possesses a natural immunity to it.

Symptoms — In some cases the course of the disease is so rapid that affected fowls die without exhibiting any symptoms. As a rule, however, the course of the infection is from two to seven days, during which time definite symptoms appear. The disease is first evidenced by depression and droopiness. The affected bird stands in one place with head drawn in and eyes closed, and is not easily roused. A staggering, irregular gait is observed when the bird is made to move. Difficult respiration is often seen and there is a wheezing or rattling sound during breathing caused by the accumulation of mucus in the nostrils and pharynx. The head is shaken at intervals in an effort to dislodge the sticky exudate which has gathered in the nostrils. In the early stages there may be an accumulation of frothy exudate in the mouth and throat which drips out of the beak in considerable quan-

tities. The nasal discharge is also profuse at times and, because of its extreme infectiousness, is an important factor in disseminating the disease.

As the disease progresses, the sick fowl is no longer able to stand and assumes a recumbent position, with prostration and death soon ensuing. The comb and wattles are bluish-red in color and swollen. The swelling often extends to the eye region, and may in some cases involve the entire head. Inflammation of the eye membranes is sometimes observed and is accompanied by excessive tear production, while in other cases these symptoms are absent. Involvement of the nerves is indicated by a shaking movement of the head, or in some instances by paralysis of the legs. Diarrhea is only occasionally observed. In some instances there is a dark discoloration of the skin and whitish scales may be found on it as well as on the comb and wattles. Recovery rarely occurs. Death may take place within a few hours, or the affected bird may linger as long as six or seven days.

Postmortem Appearance — In those peracute cases in which death has occurred suddenly with no symptoms manifested, the autopsy findings are frequently negative. In less acute cases a pronounced accumulation of sticky mucoid exudate is noted in and around the nostrils, also in the mouth.

The presence of bright red petechial hemorrhages in several parts of the body is somewhat characteristic of fowl plague. They may be found in the subcutaneous tissue, in the larynx and trachea, on the under surface of the keel bone, under the horny lining of the gizzard and in the fat surrounding this organ, in the proventriculus, in the heart muscle and on the pericardial sac, on the outer surface of the intestines and in the abdominal fat. The hemorrhages in the proventriculus and on the under side of the keel bone are constant and constitute an important diagnostic finding.

Catarrhal inflammation of the intestines is observed in some instances, but when present is usually confined to the upper part of the tract. The liver, pancreas and spleen show no constant changes, but the kidneys are usually swollen and congested. Occasionally in hens with functional ovaries, the blood-vessels of the larger ova are markedly injected and full, while in male birds swelling and petechiation of the

testes are sometimes seen. Congestion and pneumonia are not infrequently observed and there are hemorrhages on the pleural membranes. The presence of a gelatinous exudate, dotted with hemorrhages, under the skin is often noted and is regarded by some as being practically pathognomonic.

Diagnosis.—The extreme acuteness, high death-rate, and the finding of a generalized hemorrhagic condition at autopsy suggest the presence of fowl plague. Peracute cases of fowl cholera, as well as laryngotracheitis might be confused with fowl pest, but the lack of extensive hemorrhagic involvement in the former and the usual confinement of the lesions in the latter to the respiratory tract, aid in differential diagnosis.

Transmission.—Contamination of the feed, water, utensils and shipping crates by the highly infectious nasal and mouth exudates, as well as by the droppings of diseased fowls, serves as the principal means of communicating the infection. The virus might easily be carried on the clothing of persons and on feed bags or other articles brought from premises on which the disease has occurred. Wild flying birds might readily transmit the virus, and the fact that many such birds are susceptible to the infection renders their potential rôle in this connection more probable than if they were merely carriers. Shipment of fowls in the incubative stage of the disease might easily serve as a means of disseminating the virus and thus enable new foci of infection to be established.

Spread of the disease is facilitated by close contact of the birds and in the outbreak of 1925 the incidence was greatest in those places in which large numbers of fowls were kept in close confinement, particularly in the large market centers.

Ectoparasites, so far as is known, play no part in disseminating the virus. Ticks taken from a bird sick with fowl plague failed to produce the disease when crushed and injected into susceptible fowls. Beaudette, Hudson, and Saxe (1934) reported negative results from their attempts to transmit fowl plague by means of mosquitoes.

Mortality.—The death-rate from fowl plague is exceedingly high, ranging from 50 to 100 per cent.

Treatment.—Medicinal treatment is of no avail and if fowl plague appears, control and eradication should be the only measures considered.

Control—If fowl pest is suspected the proper regulatory officers should be notified at once so that control measures may be instituted immediately in an effort to limit the spread of the infection. Because of the extreme infectiousness of the disease vigorous methods are required in handling it. All sick and exposed birds should be killed and destroyed by burning. All equipment with which the diseased birds have been in contact should be thoroughly cleaned and disinfected. Compliance with regulations prescribed by Federal and State officials should be the first duty of the poultryman in the event of fowl plague making its appearance in this country.

INFECTIOUS BRONCHITIS

A respiratory disease of chicks designated as infectious bronchitis was first reported by Schalk and Hawn (1931) and later by Beach and Schalm (1933, 1936), Beaudette and Hudson (1937) and Delaplane and Stuart (1939). It is a highly infectious condition and appears to be rather widely disseminated.

Cause—The cause of the disease is a filterable virus *Tarpeia pulli*. It is interesting to note that although Beaudette and Hudson were able to propagate the virus by inoculation of the chorio-allantoic membranes of developing embryos its growth in that medium was not characterized by the appearance of gross lesions such as are seen when the viruses of fowl pox and infectious laryngotracheitis are grown by that method. This difference has also been noted by Delaplane and Stuart (1939). The virus is present in the bronchial, tracheal and nasal exudates of affected chicks and has also been found in the liver, spleen, kidneys and blood.

Occurrence—Infectious bronchitis has been reported from widely separated areas in the United States and it appears that the condition is rather common.

Susceptibility—Chickens are the only species of birds which have thus far been found to be susceptible. Attempts to infect pigeons were not successful. Early descriptions of infectious bronchitis indicated that the disease was largely confined to young chicks but subsequently it has been found that older fowls are susceptible. Beach and Schalm (1933)

reported that birds from ten to one hundred and twenty days of age were equally susceptible to artificial inoculation. Delaplane and Stuart (1939) state that the disease is found most frequently in semi-mature and adult chickens, less commonly in brooder chicks.

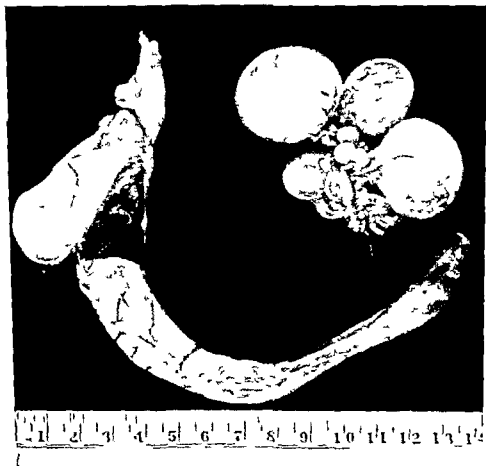


FIG. 48 — Malformed oviduct from a hen which had been exposed to infectious bronchitis virus at one day of age. (Courtesy of Minnesota Agricultural Experiment Station.)

Symptoms — The symptoms displayed by affected chickens resemble those of infectious laryngotracheitis rather closely. There is evidence of tracheitis, bronchitis with sneezing and coughing, and "crackly" breathing is often heard. A serous or catarrhal nasal exudate is usually in evidence, but there

is seldom any swelling of the face. Watery eyes are not uncommon.

Growth may be retarded temporarily in young chickens but in laying hens egg production is severely reduced. The course of the disease may be rapid with most of the birds contracting the infection and recovering within a week or ten days or it may persist for as long as three weeks. Van Roekel *et al* (1951) reported the loss of exterior egg quality as well as interior quality in laying flocks affected with infectious bronchitis. Laying of poor quality eggs may persist for weeks.

Diagnosis—The differentiation of infectious bronchitis from other respiratory diseases such as Newcastle disease, infectious laryngotracheitis and others is difficult because in the early stages the symptoms of these diseases are very similar. The isolation of the virus in embryonating chicken eggs is an important laboratory procedure. The characterization of the virus by cross serum neutralization tests and cross-immunity test is required.

Transmission—The disease is easily transmitted from infected to susceptible chickens and may spread rapidly in an area. The introduction of affected chicks or carrier birds into the flock serves as a ready means for bringing the disease into the group. In uncomplicated infectious bronchitis the symptoms may persist for one to two weeks. Hofstad (1947) showed that chickens which had recovered from field outbreaks of the disease when from one to five weeks of age were able to transmit the infection by being placed in contact with susceptible chickens for as long as thirty-five days after having recovered. Under field conditions however it appears that carrier birds exist for a much longer period.

Mortality—In older birds the losses from infectious bronchitis are not significant but in younger chickens they may be as high as 70 to 80 per cent.

Treatment and Control—There is no specific treatment for infectious bronchitis. In young chicks it is helpful to increase the temperature of the room as well as under the brooder. Anything the poultryman can do to keep up feed consumption in the flock will decrease weight loss and aid in bringing a laying flock back into production. The use of

antibiotics in feed or water has little effect on the disease but may control losses from secondary infections that often follow an outbreak of infectious bronchitis.

Vaccination programs have been extensively used particularly in the immunization of replacement laying flocks. Two types of programs have been used. Van Roekel *et al* (1950) reported on the program of deliberately inoculating 5 to 10 per cent of the flock with the virus between six and fourteen weeks of age and allowing the disease to spread to the rest of the flock. This program has limitations in that it should be used only in areas where bronchitis has been a problem. A vaccinated flock may serve as a focus of infection to set off an extensive outbreak in an area.

The second program is the use of modified live virus vaccines. These vaccines may be applied to individual birds by the intranasal or intraocular route or by mass vaccination procedures using a spray, dust or adding to the drinking water. It usually requires about three weeks for most birds to obtain a high degree of immunity following vaccination.

ORNITHOSIS AND PSITTACOSIS

There are a number of diseases of birds that are transmitted to man, but psittacosis is probably the one that has received most attention in recent years. Psittacosis is primarily a disease of psittacine birds, such as parrots, parakeets, and love birds, but over 70 or more species of birds have been found infected. Ornithosis has been accepted as a broad term to cover the infections of all birds and psittacosis has been used to designate the disease in man. In recent years the disease has been identified in turkeys, ducks, and chickens, and in some instances serious outbreaks of the disease in man have been related to handling and processing of poultry (Irons *et al*, 1951), (Andrews, 1957).

Cause—The cause of the disease is a filterable agent belonging to the rickettsial group and identified as *Mycoplasma psittaci*. It is readily cultivated in embryonated chicken eggs and experimental animals such as mice and guinea pigs. The very small organisms can be stained and observed by

is seldom any swelling of the face. Watery eyes are not uncommon.

Growth may be retarded temporarily in young chickens but in laying hens egg production is severely reduced. The course of the disease may be rapid with most of the birds contracting the infection and recovering within a week or ten days or it may persist for as long as three weeks. Van Roekel *et al* (1951) reported the loss of exterior egg quality as well as interior quality in laying flocks affected with infectious bronchitis. Laying of poor quality eggs may persist for weeks.

Diagnosis—The differentiation of infectious bronchitis from other respiratory diseases such as Newcastle disease, infectious laryngotracheitis, and others is difficult, because in the early stages the symptoms of these diseases are very similar. The isolation of the virus in embryonating chicken eggs is an important laboratory procedure. The characterization of the virus by cross serum neutralization tests and cross-immunity test is required.

Transmission—The disease is easily transmitted from infected to susceptible chickens and may spread rapidly in an area. The introduction of affected chicks or carrier birds into the flock serves as a ready means for bringing the disease into the group. In uncomplicated infectious bronchitis the symptoms may persist for one to two weeks. Hofstad (1947) showed that chickens which had recovered from field outbreaks of the disease when from one to five weeks of age were able to transmit the infection by being placed in contact with susceptible chickens for as long as thirty-five days after having recovered. Under field conditions, however, it appears that carrier birds exist for a much longer period.

Mortality—In older birds the losses from infectious bronchitis are not significant but in younger chickens they may be as high as 70 to 80 per cent.

Treatment and Control—There is no specific treatment for infectious bronchitis. In young chicks it is helpful to increase the temperature of the room as well as under the brooder. Anything the poultryman can do to keep up feed consumption in the flock will decrease weight loss and aid in bringing a laying flock back into production. The use of

lesions, pericarditis and peritonitis have been observed in affected pigeons.

In turkeys the air sacs are cloudy and show accumulation of exudate. There may be fibrinous exudate over the surface of the liver, thickened pericardium and accumulation of exudate in the heart sac and on the myocardium. Birds in production may have accumulation of egg yolk material in the peritoneal cavity.

Diagnosis.—Laboratory procedures including identification of the causative organism, the inoculation of test animals, and the use of the complement-fixation test are employed in establishing a definite diagnosis. The extreme infectiousness of the condition for both birds and humans emphasizes the necessity for expert aid if the condition is suspected.

Transmission.—Spread of the infection occurs primarily by means of body excreta. Contamination of feed and water takes place readily and in this manner transmission is effected from diseased to healthy birds. Man contracts the disease by inhaling infectious particles of dust or droplets originating with a sick bird.

Davis *et al.* (1957) reporting on egg transmission studies in turkeys, were unable to isolate the virus from freshly laid eggs or from dead embryos, or poults obtained from eggs laid by breeder hens experimentally infected with a strain of the ornithosis organism.

Mortality.—The death rate from psittacosis in psittacine birds varies greatly under different conditions. In turkeys the death rate may be as high as 100 per cent in experimental infections with virulent strains. Strains of low virulence may cause little or no mortality.

Treatment and Control.—Davis and Delaplane (1955) found that chlortetracycline in high levels in the feed prevented mortality in three-week old poults infected with ornithosis virus. Loosli (1955) has found that chlortetracycline, oxytetracycline, chloramphenicol and erythromycin were highly effective against a virus of the ornithosis group. Meyer (1955) reported on the successful use of tetracycline in the feed to control psittacosis in parakeets.

Because of the public health aspects of the problem, an early diagnosis of ornithosis is important and the outbreak should be reported to state control officials for their action.

special techniques and can pass through only the coarser grades of filters

Occurrence — In addition to its presence in the United States, psittacosis has been reported from Italy, Switzerland, France, Argentina, Austria, Denmark, Egypt and other countries and may therefore be said to be practically world-wide in distribution

Susceptibility — As has been stated, psittacosis is primarily a disease of parakeets, parrots, love birds and other members of the psittacine group, but the infection has also been identified in canaries, finches, pigeons, ducks, chickens and turkeys. Young birds are more susceptible than older ones. Human beings are susceptible and people of middle age are more likely to contract the malady than are children. Meyer (1948) stated that the infection in humans is characterized by general malaise, chills, headache, backache, anorexia, nausea, vomiting, abdominal distention, thirst, sweating and photophobia, marked constipation in most cases, restlessness, insomnia, nosebleed, cough, and an atypical pneumonia. He added that the mortality rate of 20 per cent given for humans is probably high because mild, unreported cases are possibly of frequent occurrence.

Symptoms — Birds affected with psittacosis may exhibit no symptoms at all, while in some cases progressive weakness, diarrhea, prostration and a discharge from the eyes and nose are evident. Apparently well birds may harbor the infection and be capable of transmitting it to susceptible subjects. These unrecognized, latent infections in aviaries and breeding establishments may range from 10 to 90 per cent, and when these birds are brought under unfavorable conditions of housing and nutrition, they may react and display some or all of the symptoms named above.

Turkeys affected with ornithosis show droopiness, drop in feed consumption, loss of weight, and sulfur-colored droppings. The birds may show tracheal rales and there will be a drop in egg production.

Postmortem Appearance — Emaciation, splenic tumor, and an enlarged, saffron-colored liver studded with wedge-shaped pale areas may be found at autopsy. In addition to the above

Treatment.—Wills and Delaplane (1955), and Olson *et al.* (1957) have reported that chlortetracycline and oxytetracycline reduce the mortality and delay the occurrence of synovitis. For the drugs to be effective they should be used before the onset of symptoms. After individual birds become affected the treatments appear to have little practical value.

AVIAN ENCEPHALOMYELITIS

This disease of young chicks was first described by Jones (1932) and designated by her as "an encephalomyelitis in the chicken." The same author later applied the term "epidemic tremor" to the condition but workers who subsequently have studied the disease have referred to it as avian encephalomyelitis. The determination that a filterable agent is causative adds the disease to the growing list of poultry maladies which are virus-caused.

Cause.—Jones (1932, 1934) suggested that avian encephalomyelitis is caused by a filterable virus and the fact has been well established by Van Roekel, Bullis and Clarke (1938, 1941), Olitsky (1939) and Jungherr and Minard (1942).

Occurrence.—Avian encephalomyelitis has been reported from most of the New England states, also in New York, New Jersey, Delaware, Virginia, Indiana, Colorado, Georgia, Tennessee, and Florida, and it is quite probable that it occurs unrecognized in other sections of the country. It has also been found in Australia. In the outbreaks so far observed, the greatest incidence has been in the late winter months with a few sporadic outbreaks appearing during the summer.

Susceptibility.—Chickens appear to be the only species of birds naturally affected, and the disease has been reported only in young chicks with susceptibility being greatest in birds from one to three weeks of age. The infection has been successfully transmitted experimentally to young pigeons, turkey poults and ducklings, but attempts to infect mature pheasants and sparrows were not successful.

Symptoms.—The condition has been observed in chicks as soon as 24 hours after hatching and natural outbreaks usually occur within the first two weeks of life. Affected chicks usually show a rapid vibration of the head and neck, and body

INFECTIOUS SYNOVITIS

This disease was first reported in 1954 by Wills in Texas and Olson in West Virginia. It is an infectious disease of chickens and turkeys characterized by involvement of synovial tissues.

Cause — The causative agent has not been characterized but has certain properties of a large particle virus. It has been grown in the developing chicken embryo and tissue culture.

Occurrence — The disease has been recognized in the major broiler raising areas of the United States and probably exists in all sections.

Susceptibility — Chickens and turkeys are susceptible to the agent and the disease may be reproduced experimentally by various routes of inoculation. Young birds four to ten weeks of age appear to be most susceptible. The incubation period is from eight to ten days.

Symptoms — The birds develop lameness with swelling of tendon sheaths and joints. They become listless, lose weight and dehydrate because of reluctance to walk. The droppings may be blue-green in color. The sternal bursæ become enlarged.

Postmortem Appearance — Nearly all the joints may be found involved. A mucoid exudate is present in the joints. The liver appears swollen with greenish discoloration and the spleen is enlarged.

Diagnosis — Because there are other diseases that may produce involvement of joints and internal organs, the diagnosis must be based on the isolation of the causative agent in embryonated chicken eggs or by bird inoculation. The disease must be differentiated from staphylococcal arthritis.

Transmission — The disease can be reproduced experimentally by various routes of inoculation. There is little information on how the disease spreads naturally. Contact experiments have been successful. Wills and Delaplane (1955) studied the possibilities of egg transmission with inconclusive results.

Mortality — The mortality is usually low but a high percentage of the birds may show signs of the disease.

the causative virus, viz.; Eastern and Western: the former being found east of the Appalachian mountains and the latter west of that range.

Beaudette (1939), Van Roekel and Clarke (1939), Beaudette, Black, and Hudson (1941), and Sellards, Tyzzer and Bennett (1941) have reported finding the virus (Eastern type) in naturally infected pheasants. It has also been shown by Van Roekel and Clarke (1939) that the common English sparrow is highly susceptible when artificially inoculated and young chicks have also been infected experimentally.

The exact means by which the disease is spread among pheasants are not known, but field observations indicate that it may be disseminated by certain kinds of mosquitoes which have been shown to be capable of transmitting the virus from one animal to another experimentally.

Little is known about the extent of the disease among birds, or about the symptoms, pathology and other phases of the malady. Losses in pheasants may be heavy as indicated by the report of Beaudette, Black and Hudson (1941) who estimated that equine encephalomyelitis had caused the death of approximately 65 per cent of the birds in one pen under their observation. In another group the mortality was about 25 per cent.

The significance of this disease in birds is not known at present but if it should be found that common barnyard fowls are naturally susceptible, the malady would at once assume importance, not only from the standpoint of losses in flocks but also from the possibility that such infections might well serve as reservoirs for the already serious disease in horses.

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FOWL POX

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¹ References marked with an asterisk (*) contain carefully selected or fairly complete bibliographies.

tremors may also be detected and intensified by handling the birds. These attacks may be followed by periods of dullness, and there is a progressive ataxia or lack of coordination of the muscles of the legs, with the chicks being inclined to sit on their "haunches." When made to move they have little or no control over their movements. At first the birds are in good condition but if the ataxia is marked they are unable to reach food and water, and rapid loss of weight and death ensue.

Postmortem Appearance — No gross lesions are in evidence at autopsy although microscopic changes may be demonstrated by suitable laboratory technique.

Diagnosis — The occurrence of the symptoms listed above in young chicks is suggestive of the presence of avian encephalomyelitis.

Transmission — The exact means by which the disease is transmitted are not known but spread has been accomplished experimentally by contact and it is probable that this also occurs under natural conditions. Van Roekel, Bullis and Clarke (1941) were able to produce the disease in young chicks by inoculating the eggs prior to hatching, which points to the possibility of transmission through the egg. Field studies also tend to bear out this possibility.

Mortality — The losses from avian encephalomyelitis show a wide variation, ranging from 3 to 67 per cent. Jungherr and Minard (1942) state that where symptoms occurred during the second week of brooding, the mortality was very high.

Treatment and Control — At present no measures are known to be effective in controlling avian encephalomyelitis. Until additional knowledge regarding the disease is available, sanitation must be regarded as the only sound procedure in attempts to handle the condition.

EQUINE ENCEPHALOMYELITIS

It not only is of interest, but of potential importance that certain birds have been found susceptible to infection with the virus of equine encephalomyelitis. This is a serious disease of horses and there are two immunologically different strains of

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intestinal type in older fowls. The various species of coccidia found in some of the common birds are as follows:

*Chicken:**Eimeria acervulina**Eimeria brunetti**Eimeria hageni**Eimeria maxima**Eimeria mitis**Eimeria necatrix**Eimeria praecox**Eimeria tenella**Duck:**Tyzzeria perniciosa**Goose:**Eimeria truncata**Turkey:**Eimeria adenæides**Eimeria dispersa**Eimeria gallopatornis**Eimeria innocua**Eimeria meleagridis**Eimeria meleagrimitis**Eimeria subrotunda**Quail:**Eimeria dispersa**Pheasant:**Eimeria phasiani**Pigeon:**Eimeria labbeana*

With very few exceptions, the organisms are host-specific; i. e., they are infectious for but one species of fowl.

The life histories of the coccidia are complicated, but the cardinal points of the cycle of reproduction are essentially the same for all species.

(a) *Asexual Cycle*.—The oöcyst, which is the largest and most resistant stage of the organism and the one most readily detected by microscopic examination, is passed out with the droppings of the infected fowl. These oöcysts are not yet infective but, under favorable conditions of warmth and moisture, they undergo development (sporulation) within twenty-four to seventy-two hours with the formation of sporocysts. These sporulated oöcysts (sporocysts) are infective if ingested by a susceptible bird. Development continues after ingestion, with the formation in the sporocysts of small, spindle-shaped bodies called sporozoites. In the bowel these sporozoites are released from the oöcyst shell and enter the epithelial cells of the intestine where they continue to develop and produce forms known as schizonts. As the cycle continues, the schizonts give rise to numerous small, spore-like forms called merozoites. The development and activity of the merozoites in the epithelial cells of the

Chapter 8

PROTOZOAN DISEASES

Two of the most destructive diseases of poultry, coccidiosis and blackhead, as well as several less important pathological conditions are caused by various minute, one-celled organisms known as protozoa. These microscopic forms, the smallest in the animal kingdom, are found abundantly in Nature. Many of them are entirely harmless, but others are capable of causing disease in the higher animals. Poultrymen and turkey growers are all too familiar with the ravages of coccidiosis and blackhead, the losses from these two diseases being widespread and relatively enormous.

COCCIDIOSIS

Coccidiosis is probably the most prevalent infectious disease confronting the poultry industry, and as such it has been the object of wide study and experimentation. It is a protozoan disease of the intestinal tract of chickens and other birds, usually subacute or chronic in its course, but sometimes acute in its manifestations. It is most injurious to young chicks, although older fowls are often seriously affected. In rare cases the disease in geese assumes an unusual and highly fatal form in which the kidneys are the organs principally involved.

Cause.—Small protozoan parasites called coccidia are the cause of the disease. They are found in the intestinal and cecal walls, as well as in the bowel contents of affected fowls, and their location in a particular part of the tract serves as an aid in determining the species to which they belong. It was formerly believed that all coccidiosis in fowls was caused by a single species of the organism, but it is now known that there are at least eight species capable of infecting chickens alone. Two of these are extremely pathogenic; *Eimeria tenella* producing the cecal type of the disease in young chicks, and *Eimeria necatrix* being the cause of the chronic

Occurrence.—Coccidiosis occurs wherever poultry is raised and it is prevalent in flocks all over the world. Because of the favorable conditions offered for development of the oöcysts during the spring and summer and also because young, highly-susceptible chicks are being reared at that

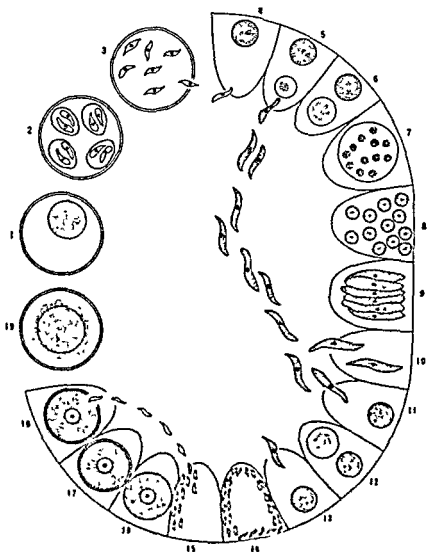


FIG. 49.—Diagrammatic representation of the life cycle of coccidia. 1, oöcyst; 2, sporulated oöcyst; 3, liberation of sporozoites; 4, sporozoites entering epithelial cells; 5-11, schizogony: formation of schizonts and merozoites; 12, sporogony, formation of macrogametocyte, 13-15, sporogony, formation of microgametocytes, 16-17, development of macrogametocyte; 18, fertilisation; 19, formation of oöcyst. (After Morgan and Hawkins, 1915.)

intestine result in the destruction of these cells and the liberation of the merozoites into the intestinal canal. From here they enter new cells with the subsequent formation of more schizonts. This schizont-to-merozoite-to-schizont phase of the life cycle is repeated several times, resulting in severe damage to the intestinal mucous membrane.

(b) *Sexual Cycle*—After several generations have been produced by the asexual cycle (without male and female forms) a change occurs in the cycle which is characterized by the formation of male and female cells. These forms, instead of more schizonts, arise from the merozoites in the epithelial cells. Soon after the appearance of these sexual cells in the intestinal contents, there is union of the male and female forms to produce oocysts. After being passed out with the droppings, the oocyst normally sporulates and, upon being ingested by a susceptible fowl, the cycle is continued.

The time necessary for completion of the life cycle varies, but under average conditions it is from four to ten days. The time required for sporulation of oocysts also varies, but the very fact that it does require some time after being passed in the droppings is an important point in connection with any attempts to control the infection.

Sporulated oocysts are quite resistant to external influences and have been found to exist in a viable state for as long as six months in the soil of poultry lots. Delaplane and Stuart (1935) found oocysts surviving for as long as eighteen months in the soil of a wooded range following removal of all chickens. Extreme dryness is destructive to them, as is excessive heat, and cold is detrimental to their development.

Working with *E. tenella*, Ellis (1938) found that high humidity favors viability of the oocysts while low humidity retards their development. He also determined that the optimum temperature for sporulation of the oocysts is between 80° and 90° F. Sporulation did not occur at 40° F or at 98.6° F and above, but viability was not, however, destroyed at these temperatures. Putrefaction of the matter in which oocysts are contained also destroys them. Disinfectants in the strengths ordinarily employed do not kill the oocysts, although continued contact with strong solutions of such disinfectants will eventually destroy them.

apparently eat but little, the crops are often full. Despite this, emaciation follows and many of the birds die. In the later stages of the disease there is paleness of the comb and wattles. The greatest losses occur within six to ten days following the onset of symptoms, although some infected chicks may linger for weeks before they finally succumb. A varying percentage of diseased chicks may recover, but if infection has been severe the survivors are usually unthrifty and therefore unproductive.

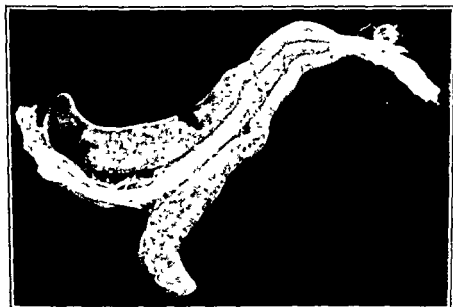


FIG. 50 —Enlarged hemorrhagic condition of the ceca, characteristic of the presence of one species of *Eimeria*, the causative organism of coccidiosis. (Courtesy of Illinois Agricultural Experiment Station)

In older fowls the chronic intestinal type of the disease is most common. Its development is sometimes slow, with only a few birds in a flock showing evidence of infection at one time. Paleness of comb and wattles; gradual loss of appetite; progressive emaciation; ruffled, dirty feathers; and disinclination to move are symptoms frequently observed. Diarrhea may or may not be in evidence. Leg weakness is commonly manifested and in some cases is so severe that the affected fowl may be practically paralyzed. Birds so afflicted are often spoken of as suffering from "range paral-

time, the incidence of the disease is greater during those seasons than at other times of the year. Outbreaks are more likely to occur during periods of wet weather. Chicks from three weeks to three months of age are readily susceptible to coccidial infection, and it is in fowls of this age that the severest losses occur. Older fowls are frequently affected and in them the disease more often assumes a chronic form. As a result of his work Mayhew (1934) found that an age immunity or resistance to coccidial infection is not acquired since typical cases of the disease were observed in birds inoculated at various ages between four and forty weeks. In contrast to the results obtained by Mayhew, it is stated by Herrick, Ott, and Holmes (1936) that chickens infected when three months old or older are considerably more resistant to the effects of *E. tenella* than are chickens infected when not over two months old.

Gardiner (1955), working with *E. tenella*, inoculated parasite-free chicks at weekly age intervals of one through six weeks with 50,000, 100,000 and 200,000 sporulated oocysts per chick. He found the four-week old chicks least resistant. One-week old chicks were resistant to all but the largest numbers of the organism. The studies of Brackett and Bliznick (1952) included chickens up to twelve weeks of age. They reported that young birds were more severely affected than older ones following the ingestion of equal numbers of oocysts of *E. necatrix*. They suggested that older birds may be less resistant than young ones if inoculation is equalized on a body weight basis.

Rosenberg and co-workers (1941, 1954) and Champion (1954) have shown that resistance and susceptibility to cecal coccidiosis are hereditary. Selective breeding was effective in establishing both resistant and susceptible lines. Survival in the resistant lines was not sufficiently high to warrant substituting this approach for other established methods of controlling the disease.

Symptoms—Chicks suffering from coccidiosis usually show droopiness and depression as the first symptoms. The chicks stand huddled, with wings dropped, feathers ruffled and eyes closed. Diarrhea is commonly present and the loose droppings are frequently mixed with blood. Although the birds

the droppings, it naturally follows that the greatest sources of infection are those places where the droppings are most abundant. In a study of the distribution of coccidial oocysts on a poultry farm, Andrews and Tsuchiya (1931) found them to be most numerous under perches and brooding canopies, in and around drinking fountains, and around feed hoppers.

To test the possibility of chickens becoming carriers of coccidia as a result of the oocysts becoming enmeshed in the tissues of the cecal pouches following infection, Herrick, Ott, and Holmes (1936) kept the members of a group of infected chickens free from reinfection and examined them from time to time for a period of one year. They state, "In this study 15 chickens infected with cecal coccidiosis were autopsied at intervals up to and including twelve months following infection and all but two harbored oocysts in the tissues of the cecal pouches. Viable oocysts were found to be passed in the cecal droppings and enmeshed in the tissues of the cecal pouches for a period of seven and one-half months." It would appear from these studies that fowls which have clinically recovered from an attack of coccidiosis may remain carriers of the disease and thus constitute a source of danger for all susceptible fowls in the flock.

The suggestion has also been made that flies are capable of transmitting the infection in their bodies, but adequate proof of this assumption has not, to the best of our knowledge, been brought forth. Delaplane and Stuart (1933) found that although oocysts could be demonstrated in the larvae (maggots) of house flies which had hatched in infected chicken droppings, they apparently were destroyed during the process of development of the larvae to mature flies. This does not of course preclude the probability of flies functioning as mechanical carriers of oocysts.

Because of the general host-specificity of coccidial organisms, the rôle played by wild flying birds in dissemination of the infection appears to be largely that of mechanical carriers. The probability of introduction by prowling animals by streams, and on the clothing of people must also be considered.

Mortality — Unless control measures are promptly instituted, the death-rate from coccidiosis in young chicks may

ysis," but such weakness sometimes accompanies other diseases and therefore is not diagnostic of coccidiosis.

Postmortem Appearance — The changes observed at autopsy in young affected chicks are usually confined to the ceca, although inflammation may also be noted in the lower portion of the intestinal canal. The ceca are swollen, dark red in color, and firmer than normal. Upon cutting the diseased pouches open the walls are found to be thickened and inflamed, and the contents may be composed largely of blood, or consist of a necrotic mass of yellowish cheesy, blood-stained debris.

In the more chronic type of the disease the lesions are found in the intestinal tract particularly in the upper duodenal region. The walls are thickened and inflamed, and small white spots are frequently visible through the outer membrane of the intestine. The lining of the bowel is inflamed and rough, and is covered with a sticky, mucous exudate. In some cases small hemorrhagic areas are seen in the lining and small flakes of clotted blood are found in the exudate.

Diagnosis — A positive diagnosis of coccidiosis is dependent upon finding one or more of the various forms of the organisms by microscopic examination of a thin film of cecal and intestinal contents. Such a preparation is easily made by placing a small amount of suspected bowel scrapings or contents upon a clean microscope slide and pressing it into a thin layer by means of a cover slip. No staining is necessary. If oocysts are present they appear as round or oval bodies having a dark, central, round portion, around which is a clear zone, the whole being enclosed by a double-lined wall.

A clinical diagnosis of the disease cannot be made with complete assurance, but the presence of the symptoms and lesions mentioned above are highly suggestive.

Transmission.—Transmission of the disease is dependent upon the ingestion of sporulated oocysts by susceptible birds although Levine (1940) and others report success in artificially transmitting it by the use of merozoites. The feeding habits of the fowl are especially conducive to this mode of spread. Since the oocysts are eliminated only in

tinued for several weeks at low levels, if necessary, without causing any undesirable effects. Coccidiosis is thus one of the few diseases of poultry for which medication can consistently be recommended. This does not mean that sanitation and good management can be neglected. On the contrary, they are important in reducing the amount and severity of reinfection, and in maintaining the general health and well-being of the flock.

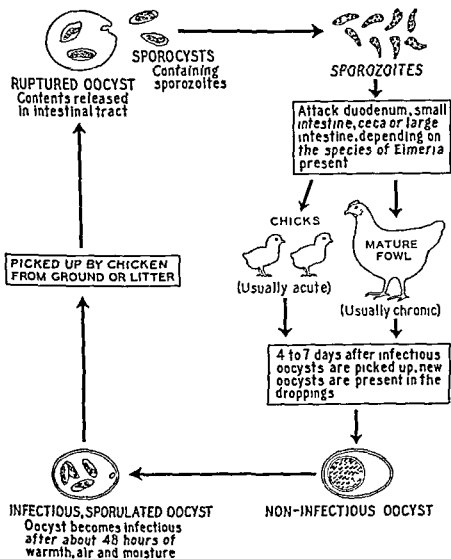


FIG. 51.—Cycle of infection in coccidiosis.

be so severe that practically all birds succumb within a short time. In older fowls the mortality may also reach a high figure, but the losses are usually distributed over a greater period of time than in chicks.

Treatment, Prevention, and Control—Research leading to the effective control of coccidiosis in chickens has involved a tremendous amount of work by many investigators, the use of a large number of drugs, and the development of management methods which included various strict sanitation procedures. Current thinking on the problem has been aptly summarized by Wehr and Farr (1956) in one sentence: "The best way to control coccidiosis of poultry is to prevent severe infections, and the best way to do that is to develop resistance to the disease."

Johnson (1927) seems to have been the first to demonstrate that a high degree of resistance could regularly be developed in chickens by experimental inoculation. A related, and highly significant fact is that resistant birds which appear perfectly healthy continue to shed oocysts which serve to infect other chickens. This led to the concept that what is needed is a drug which will control the disease to the extent of preventing a runaway infection while the chickens are developing a natural resistance. From the careful and often tedious work of such investigators as Levine (1941), Farr and Allen (1942), Wehr and Farr (1945), Swales (1946), Grumbles and co-workers (1948), Dickinson (1949), Peterson and Hymas (1950), Barber (1955), and many others, have come practical methods of continued low-level medication combined with natural infection so that coccidiosis is no longer to be feared by poultrymen who are properly informed.

It is important to remember that coccidia are almost universally found wherever chickens are raised, that repeated light infections enable the birds to build up resistance, but only to the particular species to which they are exposed, and that medication can keep the infection under control while resistance is being developed.

The drugs available commercially for use in a medicated mash include nicarbazin, nitrofurazone, nitrophenide and sulfaquinoxaline. Some are available in soluble form for administration in drinking water. Medication can be con-

susceptible to the infection, appear to possess more resistance than poults. Chickens are also subject to infection, but the disease is far less severe and not so common as it is in turkeys. The fact that chickens may harbor the organisms and yet show little or no clinical evidence of the disease is highly important from the standpoint of control. The blackhead organisms are passed out in the droppings of infected chickens, either free or enclosed within cecal worm eggs, and this constitutes a serious hazard to the highly susceptible turkey and accounts for the well known observation that the rearing of turkeys and chickens together is almost certain to result, sooner or later, in infection of the turkeys with blackhead.

The disease has also been observed in grouse, quail, pheasants, partridges and peafowls; and its occurrence in these wild birds probably accounts for its dissemination in some instances.

Symptoms.—In young turkeys the course of the infection may be so short that occasionally a bird dies without having manifested any symptoms. Usually, however, the affected poults show inappetence, increased thirst, ruffled feathers, droopy wings and drowsiness. Diarrhea is practically always present and the droppings vary from a light green to a brownish color. Durant (1937) describes orange-colored droppings as characteristic of the infection, particularly in older birds. Weakness and emaciation are evident as the disease progresses, and in some, but not all instances the skin of the head becomes dark in color.

The course of the disease in older turkeys is less severe as a rule, and progressive emaciation, sluggishness and diarrhea are characteristic symptoms. Recovery may follow an attack, but such recovered birds continue to discharge the infective organism in their droppings, and thus remain a constant source of danger to other fowls.

In chickens the manifestations may occasionally approximate those seen in older turkeys, but more often the symptoms may be so slight as to escape detection.

Postmortem Appearance.—As previously mentioned, the lesions in blackhead-infected fowls are confined to the ceca and liver. In some cases little alteration is seen in these

BLACKHEAD

Blackhead (infectious enterohepatitis) is a subacute or chronic infectious disease, in which the principal lesions are confined to the ceca and liver. It is primarily a disease of turkeys, but other fowls are sometimes affected. It is one of the most destructive infectious diseases to which turkeys are susceptible, and is responsible for large losses in the turkey-raising industry.

Cause.—The disease is caused by a microscopic protozoan organism called *Histomonas meleagridis*. These organisms are passed out of the body of an infected fowl in the droppings, and in some instances are enclosed within the eggs of the small cecal worm *Heterakis gallinæ*. Buckley, Bunyea, and Cram (1931) state that the organism seldom survives for more than twenty-four hours in an unprotected state outside the body of the fowl, but Schlotthouer, Mann, and Essex (1944) reported that *H. meleagridis* can exist in soil for a longer period. They found viable organisms in soil after the dirt had been pulverized and dried for fifteen days at room temperature. The protection given it by enclosure within the worm eggs, however, enables the organism to resist severe climatic conditions for a considerable period of time. Van Es and Olney (1934) observed for five successive years that ground left vacant by infected turkeys from the middle of November to the middle of June was still infective for four-weeks-old poults placed in the yard. Graybill (1921) had previously reported the ability of cecal worm eggs to survive exposure for a five-months period beginning in mid-winter. The fact that soil containing them was still infectious for young poults which were naturally exposed at the end of this period indicated that the blackhead organisms in the worm eggs had also resisted this severe exposure.

Occurrence.—Blackhead is widespread, being found in practically every section where turkeys are raised, and it constitutes one of the chief menaces to successful turkey raising in this country.

Susceptibility.—Young turkeys from one to three months of age are highly susceptible, and it is in poults of this age that the severest losses occur. Older turkeys, while readily

brane (peritonitis) is not uncommon in those cases in which the liver is extensively involved, and adhesion of the liver to other organs is sometimes observed.

Diagnosis.—The occurrence of a highly fatal disease in turkeys, especially poults, when manifested by the symptoms and lesions mentioned, is strongly suggestive of blackhead. Positive diagnosis is dependent upon microscopic demonstration of the organisms in smear preparations of the affected ceca. The differentiation of blackhead from coccidiosis is also possible by the use of the microscope.

Allen (1941) calls attention to the necessity of differentiating between the liver lesions in blackhead and those in trichomoniasis. Whereas the ulcer-like areas in the former disease are sunken below the level of the surface of the organ, in the latter condition these areas are irregular in outline and level with or slightly elevated above the surface of the liver. The lesions in the livers of turkeys suffering both infections are relatively large, circular, markedly depressed areas with slightly raised granular borders and sometimes with granular centers.

Transmission.—The disease is spread primarily by ingestion of the cecal worm or its eggs, both of which frequently harbor the infectious organisms. Lund (1956) states "Passage by means of the worm eggs seems to be the usual method. Thus the blackhead organism, which is fragile and can live by itself outside the bird for only a few hours and which only rarely reaches its destination in a vigorous state if swallowed alone, has in the cecal worm's egg a nearly perfect means of survival outside the bird's body."

Mortality.—In poults, the death-rate is high and may reach 80 or 90 per cent of the flock. In older turkeys the losses are not quite so severe, although the mortality may extend over a considerable period of time and involve a rather large percentage of the flock. The mortality in young chickens may be fairly high, but the number of deaths in mature chickens is usually not great.

Treatment and Control.—Many drugs have been used to treat blackhead but none has proved entirely satisfactory for stopping losses once the disease has appeared. It is true that some of the new drugs are helpful in reducing mortality,

organs, but as a rule the changes are extensive enough to be readily detected.

The lesions in the ceca may consist of raised lumpy areas in the walls of the pouches, or the entire cecum may be enlarged, inflamed and filled with a cheesy, core-like mass. The lining membrane is seriously damaged and blood is frequently found on the core of cellular debris in the gut.



FIG 52 —Characteristic lesions of blackhead in the liver. Note the difference between this and the tuberculous liver shown in Fig 32 (Courtesy of Massachusetts Department of Agriculture)

Involvement of the liver is practically always noted, and is characterized by varying degrees of enlargement of the organ, with roundish, sunken, ulcer-like areas of yellowish or salmon color over the surface. These lesions may be few in number or so plentiful as to cover the greater part of the surface of the organ. Inflammation of the peritoneal mem-

but only when combined with sound management and preventive measures. Drugs which are used to control cecal worms have no effect against the blackhead organism but by reducing the numbers of both worms and eggs may reduce the spread of the disease. The manner in which blackhead is disseminated necessitates the employment of strict measures of sanitation and hygiene if the disease is to be successfully controlled. The impossibility of detecting carriers of the organism renders it highly important that young turkeys be kept away from older birds, and that both be

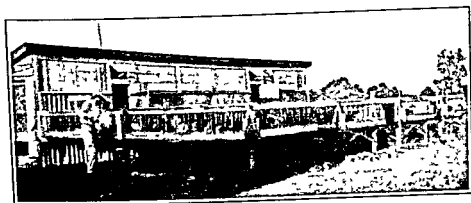


FIG. 54.—Raising turkeys in confinement on wire floors is a practical means of preventing blackhead during the early growth period. Later the turkeys are moved out to clean range.

kept entirely away from chickens and be prevented from having access to any houses, yards or ranges which chickens have inhabited.

Excellent results have been obtained by practical turkey raisers in many parts of the country by adhering rigidly to the plan of rearing turkeys by artificial means on ground over which no chickens of any age have ranged for at least two years. An alfalfa or clover field divided into four areas to be used in rotation is an ideal range for young turkeys. In addition to providing one of the requisites of effective sanitation in the form of "clean" ground, it furnishes an adequate supply of fresh green forage which is very beneficial to the growing poults.

Since it takes seven to ten days for cecal worm eggs to reach the infectious stage when conditions are favorable it



FIG. 53.—Characteristic appearance of the ceca in a turkey affected with blackhead (Courtesy of California Agricultural Experiment Station)

appears to be some degree of host susceptibility but McNeil, Hinshaw and Kofoed (1941) were able to establish the turkey organism in young quail, chicks and ducklings. The species of *Hexamita* present in quail and partridges was transmitted to poults but that from pigeons could not be established in young turkeys. Hinshaw and McNeil (1944) were unable to find *H. meleagridis* in chickens, pigeons, ducks, guinea hens, peafowls, blackbirds, English sparrows, linnets and butcher birds. Stover (1943) found a species of *Hexamita* in young ringed-necked pheasants in which heavy losses had occurred and was able to fatally infect young turkeys with material from the pheasants.

Symptoms —The symptoms displayed by affected poults are similar to those seen in some other infectious diseases and cannot be considered specific. The birds require more heat than normal, are listless and droopy, and walk with a stilted gait. The body temperature is lowered and a watery, foamy diarrhea is present. Some birds continue to eat but despite this, emaciation is rapid. The course of the disease is usually acute with death occurring in from one to seven days after the initial symptoms are noted. Those poults which survive are usually stunted and emaciated and at least one-third of them become carriers of the infection.

Postmortem Appearance —Because hexamitiasis or infectious catarrhal enteritis is primarily an infection of the upper portion of the small intestine, it is in this part of the tract that the principal lesions are found. Externally, the birds show a dry skin and emaciation. There is a marked catarrhal enteritis, the walls of the bowel are flabby and the contents are watery. There may be areas of distention in the bowel and the mucous membrane of these areas is reddened. Congestion of the glandular tissue in the region of the ileocecal openings (cecal tonsils) may also be observed.

Diagnosis —Positive diagnosis of infectious catarrhal enteritis is dependent upon microscopic demonstration of the parasites in material from the upper part of the intestine. It is necessary that such an examination be made upon a freshly killed specimen since the organisms die very quickly after the host expires.

may be necessary to move the turkey poults to a new range at similar intervals. In dry seasons or climates the interval may be considerably longer. Feeding and watering equipment should be on wire platforms so that the droppings fall through and are out of reach of the poults.

Among the newer drugs which have proved helpful in the control of blackhead are enheptin (2 amino 5-nitrothiazole) furazolidone 4-nitro-phenylarsonic acid and mthiazide [1 ethyl-3-(5-nitro-2 thiazolyl) urea]. They are very useful under what may be called emergency conditions but they should not be considered as substitutes for sanitation and hygiene in rearing turkeys.

HEXAMITIASIS

Hexamitiasis or infectious catarrhal enteritis is an acute infectious disease primarily of turkeys but which also occurs in quail and in chukar partridges. Hinshaw, McNeil, and Kofoid (1938) were the first to identify the disease in turkeys and McNeil, Platt, and Hinshaw (1939) described it in quail and partridges. Previous to these reports *Hexamita* sp. had been reported in pigeons by Noller and Buttgerieit (1923) and in ducks by Kotlan (1923). McNeil and Hinshaw (1941) reported the disease in pigeons in the United States for the first time. Before the establishment of the true nature of the disease in turkeys the condition was thought to be trichomoniasis.

Cause—A protozoan organism designated as *Hexamita meleagridis* by Hinshaw, McNeil, and Kofoid (1938) is the cause of the enteritis in turkeys and that producing the condition in pigeons is *Hexamita columbae*.

Occurrence—The disease probably occurs in all sections of the country where turkeys are raised in large numbers. It has frequently been confused with other infections partly because the organism is very small and not easily identified.

Susceptibility—Hexamitiasis is primarily a disease of poults under three months of age and it has been established that susceptibility decreases as the birds become older. Symptoms are seldom seen after the poults reach the age of two months unless unfavorable conditions lower their resistance. There

water free from contamination. The retention of old birds on the farm from one season to the next can easily furnish a source of infection for the young turkeys. Houses from which infected poults have been removed should be thoroughly cleaned, disinfected and allowed to dry before susceptible birds are put into them.

Mangrum and co-workers (1955) found furazolidone effective in the control of hexamitiasis when added to the ration at the rate of 50 mg. to the pound of feed. Lund (1956) states that aureomycin gave satisfactory results when administered at levels of 180 to 200 grams per ton of ration or in the drinking water at the rate of 10 grams of the soluble form in 50 gallons of water. Improvement was usually noted within three days.

TRICHOMONIASIS

As indicated by the name, this disease is caused by infection with protozoa known as *Trichomonas*. There are two distinct types of the condition, each caused by a different species of organism and characteristically involving different portions of the intestinal tract. The following account of trichomoniasis is adapted largely from the excellent article by Wehr and Christensen (1942).

TRICHOMONIASIS OF THE UPPER DIGESTIVE TRACT

This type of trichomoniasis involves the upper digestive tract of turkeys, chickens and pigeons, and is caused by a protozoan termed *Trichomonas gallinæ*, although the infectious agent in pigeons is termed *Trichomonas columbæ* by some authors. The disease is apparently widespread, having been reported from many sections of the country. Turkeys appear to be most frequently and severely affected.

Birds suffering from trichomoniasis show droopiness and depression, little or no appetite, sagging wings, emaciation and drooling. The disease may be very acute in young birds with death coming as soon as one day after the appearance of symptoms, while in older fowls the course is usually more prolonged in nature. In some cases the crop region is pendulous and palpation reveals a large quantity of fluid in the

Transmission.—Susceptible poults become diseased by ingesting contaminated material such as feed, soil and water. Because such a large percentage of recovered birds remain carriers, a ready reservoir of the organisms is always present where such fowls are kept. Flies have not been found to transmit the infection. Mechanical transmission on utensils and clothing must be considered as a possibility.

Mortality.—The losses in outbreaks observed so far have ranged from 20 to 90 per cent, with the greatest number of deaths occurring in poults from three to five weeks of age.

Treatment and Control.—Hinshaw and McNeil (1942) have tried a great many treatments including phenothiazine, sulfaguanidine, baking soda, and bichloride of mercury, but none was found to have any curative value and in some cases actual harm was done by the drugs.

These authors refute the opinion that the causative organisms are present on all turkey ranches and state that the source of infection has often been shown to be males purchased from infected flocks. The progress of the disease through a flock is explained by the above writers as follows: "It is seldom that the first hatch of pullets shows symptoms of the disease even though carriers are on the premises. Slight losses are sometimes experienced in a second hatch, and as the season advances each successive hatch is likely to show earlier symptoms and suffer greater losses. It works like compound interest. The first group of poults picks up a few parasites from the carriers which shed relatively small numbers. *Hexamita* multiply rapidly in poults but under ideal conditions not enough dosage is built up to cause damage before they become naturally resistant. Each successive group has additional chances to pick up the infection in larger and larger quantities, until finally acute losses are experienced."

In attempting to control the disease, therefore, any measure which will reduce the number of parasites available to susceptible poults must be employed. The temperature in the brooder should be increased and frequent cleaning without water is indicated. Segregation of the well from the sick birds is imperative as is the separation of poults from breeding stock. Especial care should be exercised to keep the feed and

sluggish, or dirty water. Hinshaw (1937) states that of the treatments tried, the substitution of a 1 to 2000 solution of copper sulphate (prepared with *soft* water and in an earthenware vessel) for the drinking water was the only one attended with any success. If the solution is used it should be kept before the birds for two or three days and the procedure repeated after a few days. During treatment, the birds should have access to no other water supply.

TRICHOMONIASIS OF THE LOWER DIGESTIVE TRACT

Involvement of the lower portion of the digestive tract is characteristic of this form of trichomoniasis as contrasted with the other type which infects the upper part of the alimentary canal. This second type of the disease is caused by *Trichomonas gallinarum*, a protozoan often found in the ceca and lower intestines of turkeys, chickens, guineas, and probably in other species of domestic birds. Although chickens are often infected, it is in turkeys that the severest infections and losses occur.

As a rule, the onset of the disease is somewhat slow. Affected birds are droopy, lose weight, and show a diarrhea characterized by yellowish fluid droppings. Young birds may die within four to eight days after the appearance of symptoms, but in older fowls the interval is much longer.

The principal lesions of this type of trichomoniasis as seen at autopsy are confined to the liver and ceca. The liver lesions are irregular in shape, usually elevated above the surface of the organ, cheesy, and granular in appearance. Although these changes superficially resemble the liver lesions of blackhead, careful examination enables a differentiation to be made. Similar lesions may be present on the walls of the ceca, and one or both of these pouches may be filled with cheesy masses of tissue, infiltrated with blood.

Diagnosis of the condition is dependent upon microscopic demonstration of the parasite and by the rather characteristic lesions. Gross differentiation of this type of trichomoniasis from blackhead requires careful examination, and confirmation should be obtained from a laboratory.

organ. Affected birds may extend the head and neck and make frequent attempts to swallow.

Trichomoniasis of the upper digestive tract is characterized at autopsy by the presence of rough protruding, yellowish, raised, cheesy areas which are firmly embedded in the mucosa and submucosa of the crop and esophagus less frequently in the proventriculus. These areas may coalesce and form a continuous large lesion with complete destruction of the mucous membrane.

Diagnosis of the disease is made by microscopic examination of lesion material and droppings and by the presence of the symptoms and lesions mentioned.



FIG. 55.—A Necrotic ulceration of the esophagus and crop seen in trichomoniasis. B typical pyramidal like necrotic ulcers characteristic of trichomoniasis of the upper digestive tract. (Courtesy of California Agricultural Experiment Station.)

The mortality ordinarily is not severe although when birds are kept under unfavorable conditions losses may exceed 75 per cent of the flock.

Transmission probably occurs by means of contaminated feed and water and since some recovered birds may become carriers of the infection it appears that they serve as a reservoir of recurrent outbreaks.

Because the condition appears to be spread principally by insanitary conditions the importance of sanitation should be stressed. All fowls especially turkeys should be kept away from areas in which they might have access to stagnant

as many as six treatments were required in more advanced cases. Of a group of 24 birds treated, 20 recovered and 4 failed to respond. Recovered birds became active, had good appetites and gained weight. Examination of successfully treated birds showed the liver lesions to be in various stages of healing.

As is the case in dealing with other infectious diseases, sanitation about the premises is important especially as it pertains to cleanliness of the feed and water.

LEUCOCYTOZOON INFECTION

This is an acute, highly fatal disease, primarily of young ducklings and turkeys, characterized by the fact that the causative protozoan parasites invade the circulatory system of the victims and there do great damage to the blood cells. Wickware (1915) described the condition in ducklings and it subsequently has been reported in both young ducks and turkeys by several workers. Clarke (1934) reported a high mortality in young grouse in Canada as apparently associated with infection by leucocytozoa.

The infection is caused by a protozoan parasite with the organism found in ducks being designated as *Leucocytozoon simondi*, and that in turkeys as *Leucocytozoon smithi*. The disease has been reported from several sections but appears to be more prevalent in the southeastern part of the country than in other areas.

Susceptibility is greatest in turkey poults under three months of age and in ducklings from two weeks to two months old. The symptoms and lesions in both species are practically identical and there is no need to differentiate the two infections from this point on in the discussion.

The infection is acute and the symptoms may be shown for only a short time. Affected birds manifest inappetence, droopiness, weakness, and a tendency to sit down. Some birds lie prone with the head and neck extended and the wings spread out from the body. Thirst is increased and breathing rapidly becomes labored. Even though the affected birds appear drowsy, if made to move they become greatly excited, show loss of equilibrium and may go into convulsions followed

Mortality figures for the disease are not available, but it is probable that it causes rather severe losses. Because it is more chronic in nature than the disease of the upper digestive tract, and also because it resembles blackhead in some respects, it is possible that losses caused by the disease may have been erroneously attributed to the latter condition.

So far as is known transmission is direct, with the infection being spread by contaminated water and feed.



FIG. 56—Necrotic ulceration of the proventriculus often seen in cases of trichomoniasis. (Courtesy of California Agricultural Experiment Station.)

Olsen and Allen (1940) have attained success in treating the disease by use of fever therapy. Naturally infected turkeys from three to eighteen months of age, in which trichomoniasis had been definitely diagnosed were used. The turkeys were placed for one to two hours in a thermostatically controlled cabinet in which the internal body temperatures of the birds were raised from 2° to 6° F above normal. This was done by maintaining the temperature of the chamber at approximately 104° F with a relative humidity of 60 to 70 per cent. Three treatments at intervals of every other day were sufficient to check the disease in most instances, although

causative organism in the blood stream during the height of the disease and by the fact that it is transmitted by ticks (vectors)

Cause —The cause of the disease is a long, wavy, screw-like protozoan organism called *Borrelia gallinarum* (*Spirocheta gallinarum*)

Occurrence —Although widely distributed in many other countries, spirochetosis is seldom found in the United States. An outbreak in turkeys was reported in California in 1946 (Hoffman, Jackson and Rucker), and the organism recovered was found by Hinshaw and McNeil (1946) to have all the characteristics of *Borrelia anserina* (= *Borrelia gallinarum*). No ticks could be found, and no other vectors were incriminated.

Susceptibility —Chickens are readily susceptible to infection, but ducks and geese are only occasionally affected. Other birds such as sparrows, canaries, guinea-fowls and turtle doves have been successfully infected by artificial inoculation with blood containing the organisms. Young fowls appear to be more susceptible than older birds.

Symptoms —The disease usually begins with diarrhea, loss of appetite and sleepiness. The birds stand around quietly, the comb and wattles are pale, the feathers are ruffled, and the wings are dropped. As the disease progresses, emaciation and weakness are marked and the sick bird becomes prostrate. In those cases in which the course is somewhat prolonged, paralytic symptoms may be evidenced by loss of control of the legs and wings. Death may occur quite suddenly without the bird having shown symptoms, or the course may occupy from three to fifteen days.

Postmortem Appearance —In the more acute cases the spleen and liver are greatly enlarged and there is evidence of degeneration in the liver in the form of small, whitish, necrotic spots. Congestion of the intestines is sometimes noted and the epicardium is occasionally covered with fibrinous membranes. In the less acute cases there is emaciation, marked paleness of the organs, and the liver and spleen are smaller than normal in size.

Diagnosis —Positive diagnosis of spirochetosis rests upon the demonstration of the causative organisms in the blood.

by coma. When in complete stupor they may eliminate a foamy discharge from the mouth and nostrils.

An enlarged, congested spleen is practically always found upon postmortem examination. Emaciation and anemia are often noted and the flesh is yellowish in color. Mild congestion is usually seen in the crop, proventriculus, gizzard, and upper part of the intestinal tract as well as in the lungs, liver, and kidneys. There may be a slight increase in the amount of pericardial fluid and the heart muscles are paler than normal.

The disease can be diagnosed with certainty only by microscopically demonstrating some form of the protozoan in preparations of blood.

Attempts to transmit leucocytozoön infections by direct means have not been successful and it appears that an intermediate host is necessary. At least two species of small black-flies viz., *Simulium occidentale* and *Simulium nigroparvum* have been found capable of transmitting the parasites from the blood of an infected bird to a well subject. Johnson (1941) states that after the flies have fed upon the blood of an infected turkey, development of the parasite proceeds within the body of the fly, eventually resulting in a stage capable of causing the disease in susceptible birds when bitten by the fly.

Because of the absence of black-flies in the area, and the prevalence of lice on birds in cases studied by them, West and Starr (1940) have suggested that lice might serve as vectors. Subsidence of the disease following eradication of the lice formed the basis for their opinion. Recovered birds may serve as carriers and in the presence of suitable intermediate hosts, serve as the source of further infection in a flock.

Mortality in poults ranges from 10 to 50 per cent and in ducklings from 10 to 90 per cent.

No treatment has been found to be of any value in dealing with the infection. Control of the flies known to be capable of serving as intermediate hosts is necessary and Johnson (1939) has found that rearing turkeys in a fly-proof shelter is an effective means for controlling this as well as other diseases.

SPIROCHETOSIS

Spirochetosis is an acute or subacute, highly fatal, septicemic disease of fowls, characterized by the presence of the

and sparrows and is characterized by the presence in the muscles, particularly those of the breast, of white, maggot-like bodies. The cause of the disease is the protozoan *Sarcocystis rileyi*, and it is the cystic form of the organism which is visible in affected birds. The life cycle of the organism is not thoroughly understood. Although the muscles may be riddled with the parasites, their presence appears to cause little inconvenience to the bird and they are usually not discovered until the fowl is being dressed for food.



FIG. 57.—Mallard duck infested with *Sarcosporidia*. (Duck collected by Levi Phillips, Cambridge, Md. Photo for Dr. J. E. Shillinger, Fish and Wildlife Service, U. S. Dept. of the Interior. Quortrup and Shillinger, courtesy of Jour. Am. Vet. Med. Assn.)

of an affected fowl during the height of the disease. The occurrence of the infection is apparently limited to those areas in which vector ticks are found.

Transmission—Spirochetosis is transmitted from fowl to fowl by the tick *Argas persicus*, and Kelser (1933) states there is some evidence that the fowl mite *Dermanyssus gallinæ* may also be capable of transmitting the infection. The ticks feed upon the fowls at night and, if the blood ingested contains the organism, the bite of such a tick may be infectious for fowls for as long as six months thereafter.

Mortality—The death rate from spirochetosis is reported as varying from 40 to 100 per cent of the flock.

Treatment—Some success has been attained in the treatment of the disease by the employment of various arsenical drugs including salvarsan and atoxyl, but the practical application of such procedures is open to question. Marcos, Zaki, and Ragheb (1946) obtained no curative effects from the use of sulfonamides and penicillin but did get highly curative results with myosalvarsan and atoxyl. Carbolized tissue vaccines, as well as infection followed by treatment with arsenicals, proved to confer solid immunity. Sreenivasan and Sankaranarayan (1943) found atoxyl to be toxic especially in old fat birds, but were able to effectively protect birds against the disease by giving doses of 0.15 to 0.2 gram of a drug called sulfarsenol.

McNeil Hinshaw and Hissling (1949) in an extended series of experiments found penicillin to be effective in a single dose of 10,000 units when given intramuscularly to infected birds ranging from six to twelve months in age. Streptomycin proved to be ineffective.

Prevention—The control of vector borne diseases has been most successfully accomplished by eradication of the insects responsible for transmitting the infection, and it appears that this is the most effective form of prevention in dealing with spirochetosis.

SARCOSPORIDIOSIS

Although this disease is encountered frequently in mammals, it is found only occasionally in birds. It has been reported in several instances in wild ducks as well as in chickens.

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MALARIAL INFECTIONS

Several species of birds, including pigeons, mourning doves and quail, have been found to be infected with avian malarial parasites of the *Hemoproteus* group. O'Roke (1930) found the infection in California valley quail and noted a variation in the severity of the disease. In the mild, chronic type no symptoms were shown and the infection could be diagnosed only by microscopic examination of the blood. In the mild, acute type the birds were restless and off feed for two to four days, when the infection was either thrown off or became chronic. In the moderate chronic form of the disease, the birds were anemic and weak, and the author states that the infection was probably a contributing factor to death under conditions of exposure or of breeding exhaustion. In the heavy, acute type the birds lost flesh rapidly, were unable to fly, refused food, became droopy, and some died.

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by which these objectives can be attained, and it is a mistake to assume that there is any one "best" ration.

Experimental trials, biological tests and laboratory procedures will continue to be of fundamental importance in adding to our knowledge of the nutrient requirements of animals, and of the nutritive value of various feeds and supplements, but some degree of judgment should be used in applying the results to practical feeding operations.

PATHOLOGICAL EFFECTS OF FAULTY NUTRITION

Certain abnormal conditions met with in avian pathology are the result, not of specific organisms, but of a lack of some necessary constituent of the ration; of an improper balance between different nutrients; or, less frequently, of a decided excess of some food substance or nutrient. The best of sanitary measures will accomplish little unless the flocks concerned are so fed that there is no shortage of any essential nutrient. When fowls are either qualitatively or quantitatively undernourished, they become more readily susceptible to the attacks of parasites and disease organisms than they are under conditions of optimal nutrition.

DEFICIENCY DISEASES

The group of nutrients known collectively as the vitamins is of special interest because they are needed in such small amounts, and because a deficiency may have very pronounced effects on the chicken.

A detailed discussion of the vitamin requirements of fowls is, of course, beyond the scope of this book, but brief consideration will be given to those specific pathological conditions which are known to be brought about by vitamin deficiencies.

A practical difficulty is that acute symptoms which are easy to recognize, and which form a basis for accurate diagnosis, seldom occur unless the rations being fed are markedly deficient in the vitamin under consideration. Subacute or chronic deficiency symptoms are much more common, are often more serious simply because they go too long unrecognized, and are so much alike for several different de-

Chapter 9

NUTRITIONAL DISORDERS

WHEN formulating practical rations, it is important to remember that modern methods of management have made it increasingly necessary to supply fowls with sources of all of their nutritive requirements. A small flock of hens running at large can pick up many of the proteins, minerals and vitamins which are needed to supplement a grain ration. This is not possible when they are confined indoors, especially if the flock has been carefully selected and is so managed as to lay at a high rate throughout the year. Similarly, the actual nutrient requirements of rapidly growing broilers are much higher than those of range-grown chickens which are increasing in weight at the slower rates long considered normal.

The importance of proper nutrition in maintaining the health of poultry is much more commonly appreciated now than formerly, and more exact information is constantly becoming available for the use of persons who are striving to provide adequate diets for the flocks under their care. It is unfortunately true, however, that there is too often a tendency to emphasize the latest discovery, or the most recently publicized experimental result, at the expense of some of the well-established but less spectacular principles of successful feeding.

It is relatively easy to set up experimental conditions under which some minor dietary factor can be shown to be of great importance to the well-being of the animals under observation, but it does not necessarily follow that all flock owners should at once proceed to add some source of that particular factor to the rations which they have been using under practical conditions. Rations must be complete with respect to the various dietary essentials, and they must be reasonably well balanced if profitable results are to be obtained from their use, but there is usually a variety of ways

Vitamin D—Fowls of all ages have a high requirement for vitamin D. It is concerned primarily with the absorption, deposition, and retention of calcium and phosphorus. If it is deficient or absent, young chicks will soon develop rickets, a condition in which the growing bones fail to calcify normally. The chicks are unthrifty, retarded in growth, and become lame.

The development of rickets is also influenced by such factors as rate of growth, the percentage of protein in the ration, the ratio of calcium to phosphorus, and the presence of certain other minerals in the ration fed. In effect, there is a certain balance which must be maintained among these factors if maximum growth and normal bone development are to occur simultaneously.

In laying hens a deficiency of vitamin D will result in thin shelled eggs and a greatly reduced hatching power. Egg production will eventually cease.

Decker and McGinnis (1947) observed that a deficiency of vitamin D caused a widespread deposition of black pigment in the feathers of Buff Orpington chicks. Immature feathers began to show normal color at the base about five days after the ration was corrected.

Although exposure to direct sunlight will enable chickens to manufacture their own vitamin D, it is doubtful whether the needs of flocks which are laying at an extremely high rate or of rapidly growing chicks can be fully met in this way.

It is considered sound practice to add some source of this vitamin to the ration. Biologically tested vitamin D oils are readily available, and irradiated sterols in dry form are effective and economical sources.

Vitamin E—Vitamin E deficiencies in young, rapidly growing chicks may result in encephalomalacia or "crazy chick disease" and exudative diathesis. Ducks under similar conditions show muscular dystrophy, while turkey poults often show enlarged hocks and lameness. Onset of the disease is rapid with ataxia, head retraction and spasms of the limbs as prominent symptoms. Complete prostration usually follows. At autopsy the gross lesions are brain disintegration, hemorrhage and edema.

iciencies that correct diagnosis is difficult, and often impossible

Vitamins are commonly divided into two groups, fat-soluble and water-soluble. In neither group, however, do the various vitamins have much else in common beyond the fact that they are absolutely essential to normal health and production performance.

The four fat-soluble vitamins are commonly designated by letters—A, D, E and K. All but the most recently discovered water-soluble vitamins have been given names which are descriptive of their chemical nature—thiamin, riboflavin, niacin (nicotinic acid), pantothenic acid, pyridoxine, biotin, pteroylglutamic acid (folic acid), inositol, p-aminobenzoic acid, ascorbic acid and choline.

Vitamin A—In chicks deprived of vitamin A, the symptoms of deficiency begin to appear in about three weeks. Growth is markedly retarded, and there is general unthriftiness, with ruffled plumage, a staggering gait, and a disinclination to move about. If the deficiency is not corrected, mortality will reach 100 per cent within a short time.

In older fowls the symptoms develop much more slowly. The protective mechanism inherent in the mucous membranes of the entire respiratory tract is seriously damaged or even entirely destroyed so that secondary infections are frequently found. There may be a cheesy exudate from the eyes and a sticky discharge from the nostrils. Egg production and hatchability are much reduced.

Lesions seen at autopsy consist of whitish pustules in the roof of the mouth and along the esophagus, and whitish urate deposits in the kidneys and ureters. The ureters are sometimes greatly distended.

Deficiency symptoms in poults are similar to those in chicks but, because of the greater susceptibility of this species to vitamin A deficiency, any lesions observed are usually less pronounced.

Common sources of vitamin A are yellow corn, alfalfa, clover, kale and pasture grasses. Properly dehydrated alfalfa leaf meal is often an excellent source, as are certain fish oils and fortified feeding oils.



Fig. 25 — An advanced case of nutritional rickets caused by severe vitamin A deficiency
(Courtesy of California Agricultural Experiment Station)

Experimental production of the disease has depended in part on the use of cod liver oil or other fish oils as dietary stress factors. Antioxidants such as DPPD will offset the effect of the stress factor and lessen the destruction of vitamin E, thus lowering or preventing the incidence of encephalomalacia.

Vitamin E is essential for normal hatchability of both chicken and turkey eggs. The requirement has been reported as 20 mg. of alpha-tocopheryl acetate per pound of total feed.

Vitamin K.—This vitamin must be present in order for the blood to clot normally. Chicks fed a ration deficient in vitamin K are likely to have severe hemorrhages following a bruise or injury to any part of the body, and may bleed to death from such minor injuries as those incidental to wing-banding. Mature fowls are not so easily affected, but a deficiency in the ration of breeding flocks will carry over to the chicks until such time as they have been able to build up normal stores of their own. The vitamin is abundant in alfalfa meal, green pasture grasses, meat scrap, and fish meal.

Hemorrhaging in young chicks is easily produced by feeding simplified rations low in vitamin K. The hemorrhages occur in various parts of the body, especially in the breast and leg muscles, in the meninges of the brain, and under the skin of the wings. The condition is aggravated by certain drugs and it is important to supply optimum levels of vitamin K when the stress of drug medication is present. Coccidiostatic drugs should not be fed in excess of the minimum amounts needed for control of the disease. A minimum of 5 per cent of alfalfa meal is suggested as a safety factor. Added vitamin K as menadione or menadione sodium bisulfite will provide further insurance.

Cover, Mellen and Gill (1955) made extended studies of the pathology and hematology of the syndrome found in both field and laboratory cases of avitaminosis K and concluded that the two are definitely distinct and dissimilar. The etiology of field cases of hemorrhagic disease remains obscure.

Thiamin.—A ration deficient in thiamin (formerly called vitamin B or B₁) is inadequate for growth. If such a ration is fed to either young or old fowls it will cause paralysis of

hatching power. Marked improvement can be brought about by supplementing such rations with dried liver, autoclaved yeast, dried skim milk, or green forage.

It is important to remember that the riboflavin requirement for hatchability is considerably higher than for egg production and normal health of the hen. Rations which are only slightly deficient may result in high embryonic mortality especially during the second week of incubation.

Synthetic crystalline riboflavin and riboflavin mixtures are available commercially at low cost, so that rations can easily be fortified even if the usual natural sources are not available.

Niacin (Nicotinic Acid)—A deficiency of niacin in the ration of young chicks will cause retarded growth and poor feathering. A severe deficiency in ordinary rations is unlikely, but when experimentally imposed it will cause a dark inflammation of the tongue and mouth cavity. Rations high in corn, especially if they also contain bone from meat scrap, or bone meal, are very likely to be deficient in niacin and therefore incapable of producing rapid growth. Under such conditions the amino acid tryptophane will be broken down to furnish niacin, thus further aggravating a bad condition. Niacin additions are therefore indicated for high corn rations, especially for young chickens. In synthetic form it is relatively inexpensive. Good natural sources are liver, yeast, wheat bran and middlings, and most cereal grasses.

Pantothenic Acid—Young chicks fed a ration deficient in pantothenic acid show slow growth and extremely ragged feathering. Scabby lesions appear at the corners of the mouth, on the edges of the eyelids, and around the vent. In severe cases they are also seen on the bottoms of the feet. A deficiency in the ration of breeding hens results in lowered hatchability. Among the best sources are milk products, yeast, liver, cane molasses, alfalfa meal and green pasture grasses.

Pyridoxane—Continued deficiency in chicks results in jerky movements, aimless running about, followed by convulsions, complete exhaustion, and death. In mature fowls there is loss of appetite, followed by rapid loss of weight, and death. Partial deficiency causes lowered egg production.

the peripheral nerves or polyneuritis. The chicken has a high and continuous requirement for this vitamin but, since it is abundant in the grains, in the fresh green leaves of many plants, and in milk products, deficiency symptoms are rarely seen under practical conditions.



FIG. 59.—Cheesy material in the eye of a fowl affected with nutritional roup, the result of vitamin A deficiency. (Courtesy of California Agricultural Experiment Station.)

Riboflavin.—A lack of riboflavin (formerly known as vitamin G) results in very slow growth, diarrhea, and a characteristic curled toe paralysis of the legs. Some chicks may walk on their hocks, with the toes curled inward, and otherwise appear to be in excellent health.

Many rations fed to laying hens may be partially deficient in riboflavin, with the result that the eggs have low

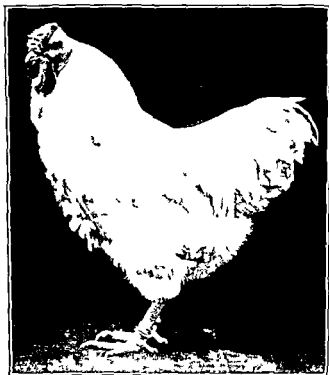


FIG 61 —The same cockerel shown in Figure 60, after one week on a diet of white corn germs grit and water (Courtesy of C W Carrick)

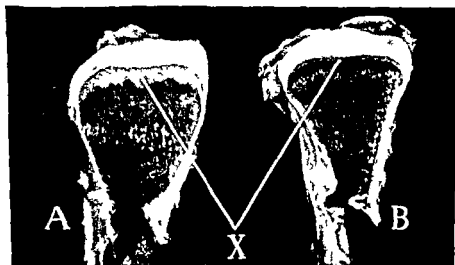


FIG 62 —The difference in calcification of leg bones of chicks with and without a source of vitamin D in the diet. Chick A was deprived of vitamin D. Chick B was fed the same ration plus $\frac{1}{2}$ of 1 per cent of sardine oil. Note the difference in the extent of the uncalcified areas, shown at X. (Courtesy of F. E. Booth Company, Inc.)

and poor hatchability. A deficiency in ordinary rations is very unlikely.

Biotin.—A deficiency in the ration of young chicks results in lesions quite similar to those observed in pantothenic acid deficiency. The bottoms of the feet become rough and caloused, and later crack open and become hemorrhagic. Eventually, lesions of a similar kind appear at the corners of the mouth, and the eyelids may become granular. The order of appearance of the skin lesions is reversed from that observed in a deficiency of pantothenic acid. Biotin is also



FIG. 60 —The result of thiamin deficiency. This cockerel had been kept for twenty-four days on a diet of degerminated white corn, grit and water. (Courtesy of C. W. Carrick.)

involved in the prevention of perosis and is essential for high hatchability of eggs. Since it is rather widely distributed in natural feeds, a deficiency is not likely under practical feeding conditions.

Pteroylglutamic Acid (Folic Acid).—Although this vitamin is required for normal growth and survival, it is particularly necessary for hemoglobin formation and the prevention of anemia. It is also essential for normal feathering. The actual requirement is very small and there is evidence that it is influenced by the type of ration fed. Recent experiments indicate that about 250 micrograms per pound of ration will cover all requirements. Chicks deprived of this vitamin for

sources has made it necessary to pay particular attention to this factor in ration formulation. Fermentation residues from the commercial production of antibiotics constitute one of the best practical sources of the vitamin.

Other Vitamins.—Inositol, p-aminobenzoic acid, and choline are all required by the chick, but since they are rather widely distributed in natural feeds a deficiency is rather unlikely. Choline, along with manganese and biotin, is necessary for the prevention of perosis (slipped tendon) in growing chicks. Since chickens are not susceptible to scurvy, there is no need to supply ascorbic acid in poultry rations. It has been shown that additions of ascorbic acid to various purified rations fed to laboratory chicks will stimulate growth to a small extent, but the general practice is to disregard this factor when formulating rations.

OTHER NUTRITIONAL DISTURBANCES

Gout.—Though not of great economic importance, gout or uremic poisoning does occur in poultry. It is a nutritional disease characterized by internal deposits of sodium urate. It is essentially a disease of mature fowls. Patterson (1928), who has given a good description of the disorder, says that it occurs most frequently among fowls kept on high-protein diets, and that any abnormality which impairs kidney function may lead to gout. Close confinement and accompanying lack of exercise are predisposing factors.

Gout nearly always runs a chronic course. It may be visceral or articular in form, depending upon whether the characteristic sodium urate deposits occur in the viscera or joints. No visceral organ is exempt. The kidneys are often enlarged, light in color, and spotted with an excess of urates. The ureters are frequently enlarged and packed with urates. The heart is often affected.

If the urate deposits are mixed with a small amount of acidulated water and examined under the microscope, numerous needle-like crystals of sodium urate will be found.

The treatment commonly recommended is to reduce the level of protein in the diet, to supply an abundance of fresh green forage, to encourage water consumption, and to pro-

the first four weeks after hatching respond dramatically to a single administration given either orally or intramuscularly. Liver is one of the best natural sources.

TABLE 4—NUTRIENT REQUIREMENTS FOR POULTRY¹
(In percentage or amount per pound of feed)

	Starting chicks	Breeding hens	Starting poult	Turkey breeders
Total protein per cent	20	15	28	15
Vitamins				
Vitamin A activity (U S P units) ²	1200	2000	2400	2400
Vitamin D (International chick units)	90	225	400	400
Thiamin mg	8	?	?	?
Riboflavin mg	1.3	1.7	1.7	1.5
Pantothenic acid mg.	4.2	4.2	5.0	?
Niacin mg	12	?	?	?
Pyridoxine mg	1.3	1.3	?	?
Biotin mg	.04	?	?	?
Choline mg.	600	?	750	?
Folic acid mg.	25	16	4	?
Minerals				
Calcium per cent	1.0	2.23 ³	2.0	2.25 ³
Phosphorus per cent ⁴	6	6	1.0	75
Salt per cent ⁵	5	5	5	5
Potassium per cent	2	?	?	?
Manganese mg	25	15	25	15
Iodine mg	5	5	?	?
Magnesium mg	220	?	?	?

¹ National Research Council Publication 301 January 1954

² May be vitamin A or pro-vitamin A

³ It is not necessary to incorporate all of this calcium in the mixed feed. Calcium supplements fed free choice are considered as part of the ration.

⁴ At least 0.45 per cent of the total feed of starting chickens should be in organic phosphorus. The same is true for poult. Approximately 30 per cent of the phosphorus of plant products is non phytic phosphorus and may be considered as part of the inorganic phosphorus required.

⁵ This figure represents added salt or sodium chloride.

Vitamin B₁₂—This vitamin is probably required in the ration by all young growing animals including the chick. It is also very necessary in the ration of breeding hens. Rations containing adequate amounts of fish meal, meat scrap and milk products are not likely to be deficient in vitamin B₁₂, but increased dependence on plant protein

vide ample opportunity for exercise. The symptoms and the remedial measures suggest a connection between gout and vitamin A deficiency, though many more fowls are affected by a deficiency of vitamin A than ever show the characteristic symptoms of gout.

Similar conditions are commonly observed in avian monocytosis or blue comb, which is discussed in the following chapter.

Perosis—Perosis, or deforming leg weakness, also called hock disease or slipped tendon, is often confused with rickets in spite of the fact that the bones are well calcified and hard, in contrast to the poorly-calcified, soft bones that characterize rickets. The legs become bowed, or badly twisted, and the Achilles' tendon slips out of its normal position. There is usually an enlargement and flattening of the hock joint, and sometimes of the entire shank.

The conditions under which perosis is most likely to be encountered in the field include the feeding of rations containing a high percentage of corn, and the addition of steamed bone meal to rations in which the chief protein supplement is meat scrap.

It is now known that several different factors may be involved in the production of perosis. Rations high in phosphorus were early found to cause trouble, especially if they were also high in protein. The condition most often results from a lack of manganese in the ration, and can usually be prevented by including 4 to 6 ounces of manganous sulfate in each ton of feed. Perosis is also influenced by a deficiency of choline or of biotin. Fortunately, neither one is likely to be deficient in normal farm rations.

Lions and Insko (1937) have shown that a diet low in manganese, which produced a high percentage of slipped tendons when fed to young chicks, was responsible for an abnormality in chick embryos known as chondrodystrophy, and greatly reduced the hatching percentage. The trouble was completely prevented by injecting small amounts of manganese directly into the white of the eggs.

Caskey, Norris and Heuser (1944) observed a chronic, congenital ataxia in newly hatched chicks caused by a manganese deficiency in the ration of breeding hens. The ataxia



FIG. 63.—“Nutritional leg-weakness” resulting from a deficiency of riboflavin (Courtesy of Cornell Agricultural Experiment Station.)



FIG. 64.—Dermatitis resulting from a deficiency of pantothenic acid (Courtesy of Cornell Agricultural Experiment Station)

vide ample opportunity for exercise. The symptoms and the remedial measures suggest a connection between gout and vitamin A deficiency, though many more fowls are affected by a deficiency of vitamin A than ever show the characteristic symptoms of gout.

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Caskey, Norris and Heuser (1944) observed a chronic, congenital ataxia in newly hatched chicks caused by a manganese deficiency in the ration of breeding hens. The ataxia

became less evident, but did not entirely disappear, as the chicks grew older. Offspring of ataxic parents were normal when both were fed a normal ration containing adequate amounts of manganese.

Briggs and Lillie (1946) observed perosis in young chicks fed 0.5 per cent thiouracil over a period of five weeks. It was not prevented by adding manganese, choline, niacin, biotin, or riboflavin to the experimental ration, and the authors suggest that thyroxine may aid in preventing perosis under normal conditions.

Anemia—Mention has been made in previous chapters of anemia as a symptom accompanying specific disease conditions. The rations commonly used for young chicks would never cause nutritional anemia but it has been observed that chicks which are kept on an exclusive milk diet become severely anemic by the time they are three or four weeks old. The condition can be relieved by the addition of iron and copper salts to the diet.

Goiter—Although unknown in many parts of the country, goiter does occur in fowls. Kernkamp (1925) has described 2 cases of simple colloid goiter which occurred in Minnesota. In one of these the thyroid was greatly enlarged, measuring 3.2×2.5 cm. Welch (1928) says that goiter in poultry is very common in Montana. The enlarged thyroid, frequently as large as the thumb, is entirely concealed by the feathers and is therefore, not commonly noticed. Welch further states that the owners of goitered flocks have always reported that egg production and general health were normal. From this it would appear that goiter does not have any important effect on the health of fowls.

Hollander and Riddle (1946) described the occurrence of many cases of goiter in pigeons reared on Long Island and in Sumter, S. C. Small supplements of potassium iodide added to the ration cured and prevented goiter in adults and in newly hatched squabs.

Attempts to show that chickens need more iodine than is obtained in ordinary poultry rations have not been successful. Hamilton and Kick (1930) in Illinois, using young White Leghorn chicks of the same initial weight and individually fed so that they received approximately the same amount of

food, were unable to find any influence on rate of growth from the feeding of potassium iodide at the rate of 0.5 or 1 mg. daily for each 100 grams of body weight. Forbes and associates (1932) in Pennsylvania fed iodized linseed meal to *White Leghorn* females from hatching time to thirty-two weeks of age, at a rate supplying 0.05 gram of iodine per 100 pounds of chicken per day, and were unable to observe any certain effect of the iodine on growth, mortality



FIG 65 —The condition known as perosis, or slipped tendon.

or egg laying. The quantities of iodine required are so very small that positive results could hardly be expected except in regions where iodine is known to be lacking in the natural vegetation and water supply.

Wilgus and co-workers (1948) studied the iodine requirements of chickens through several generations, using histological evidence of goiter as the measure of iodine need. They concluded that practical rations should contain from 0.5 to 1.0 mg. of iodine per pound.

Wilder, Bethke, and Record (1933) found that the iodine content of hens' eggs varies directly with the amount in the

ration The feeding of 2 and 5 mg of iodine daily per hen, in the form of dried kelp, iodized linseed meal or potassium iodide, increased the iodine content of the eggs approximately 75 and 150 times, respectively The amount of iodine in the eggs was independent of the form in which it was fed, but the percentage of iodine decreased immediately when iodine feeding was discontinued

Other Troubles—Several workers have observed an unusual amount of "pasting up" in young chicks when soybean oil meal is used as the chief protein supplement Berg, Bearse, and Miller (1945) reported increasing amounts of pasting up as the level of protein in a soybean oil meal ration was raised from 18 to 24 per cent The condition is likely to be aggravated by brooding temperatures which are either too high or too low

Taylor, Lerner and DeOme (1944) reported that a ration containing 53 per cent of corn when fed continuously from hatching time to the end of the first laying year, produced significantly higher laying hen mortality than a similar ration in which a large part of the corn was replaced by barley and alfalfa meal Differences in mortality were seen in ruptured and flabby ova, occluded oviducts, reverse peristalsis, and prolapse It seems probable that these troubles arose from nutritional deficiencies of corn rather than from any toxic substance contained in corn grain

Charcoal has often been recommended for its supposed beneficial effects on chicks and hens Actually it may be harmful because it can inactivate most of the vitamin A, vitamin K, and riboflavin in a chick ration

Since poultlets held without water for the first seven days after hatching had loose, slimy gizzard linings, Hammond (1944) concluded that failure of poultlets to learn to drink is the primary cause of loose, slimy gizzard linings that accompany early non-specific mortality often observed in turkey poultlets It is especially important for baby turkeys to learn to eat and drink at an early age Chilson and Patrick (1946) found that withholding feed and water for twenty-four, forty-eight, and seventy-two hours after hatching resulted in mortality to twelve weeks of age of 5, 12 and 29 per cent, respectively

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Chapter 10

MISCELLANEOUS CONDITIONS

AMONG the troubles encountered in poultry flocks are many which either are not caused by specific disease organisms or belong in a group of diseases of which the cause has not yet been clearly demonstrated

AVIAN MONOCYTOSIS

This disease of unknown cause has been called by various names, such as pullet disease, blue comb, X disease, and contagious indigestion. Jungherr and Matterson (1944) proposed the name avian monocytosis as a descriptive scientific term.

It occurs most often in pullets five to seven months of age, usually when they are in prime laying condition, but it affects birds of both sexes, has been reported in chickens no more than two months of age and occasionally in yearling or older hens. Since it often strikes without warning and affects a high percentage of the flock, it can be very costly in terms of lowered egg production. Incidence is variable from year to year and to date has been greatest in the northeastern and north central states. The death loss is usually low, 5 to 10 per cent, but may be higher. Morbidity may range from 10 to 100 per cent.

Characteristic symptoms are depression, lack of appetite, marked cyanosis or darkening of the comb and head, and a pronounced watery diarrhea. The crop is often distended with food and water which do not move into the lower digestive tract. The food in the crop soon turns sour with an offensive odor. There is usually a high degree of dehydration, shown by dry skin and thin legs and shanks. Veins in the shanks often become plainly visible.

Laying flocks drop sharply in egg production and are usually slow in recovering a normal rate of lay.

Cole (1950) found significant strain differences in mortality from this disease, with losses ranging from 0 to 7.4 per cent. He also reported significant differences between sire families within the most susceptible strain, and between dam families within sires. The data of Moultrie, Cottier and King (1955) support this view.

Postmortem appearances include a chalky condition of the pancreas, enlarged kidneys which are pale in color because of accumulated urates, distended ureters which often are clogged with crystals of uric acid, and a necrotic or spotted liver. There is usually abundant evidence of dehydration.

The blood is typically very concentrated and dark in color. The concentration makes it difficult to draw blood samples. The uric acid level of the blood is very high, and this is probably the ultimate cause of death in affected specimens.

The nature of the disease is such as to suggest that a filterable virus is probably involved, but it has not yet been demonstrated that this is the sole cause. Waller (1944, 1945) isolated a filterable agent from the blood and livers of acutely affected live fowls. A vaccine which he prepared from the dried chorio-allantoic membranes of infected embryos appeared to give some measure of protection.

There is no known specific treatment or method of prevention. Potassium treatment seems to be helpful. Since the birds usually have little appetite, treatment is best made through the drinking water. Because of the extreme dehydration, water intake should be encouraged in every way possible.

Connecticut workers reported favorable results from the addition of 2 per cent of molasses or 0.5 per cent of muriate of potash (at least 60 per cent K₂O) to the drinking water for a period of four or five days. This was followed by 1½ per cent of muriate of potash in the mash for ten to fourteen days. This treatment helps to alleviate the kidney damage caused by the disease. Other workers have found antibiotics especially aureomycin® and terramycin® to be effective when fed at levels of 100 grams per ton of feed.

BLUECOMB DISEASE OF TURKEYS

The term bluecomb disease is applied to an infectious disease that affects turkeys of all ages. It appears to be quite similar to avian monocytosis or pullet disease of chickens. It has been described as non-specific infectious enteritis, mud fever, and transmissible enteritis. The causative agent has not been completely identified but has some characteristics of a virus



FIG 66 —Emaciation and dehydration of the musculature of a bluecomb-infected poult (left) compared with a normal poult (right) (Courtesy of Minnesota Agricultural Experiment Station)

It affects turkeys of all ages. In young poults the losses may be high, from 10 per cent to as high as 100 per cent. In growing birds the loss may be low, from 1 to 2 per cent to 10 to 20 per cent. In mature fowl the loss is usually low. The disease may be encountered in all seasons of the year particularly on turkey farms that have a continuous rearing program.

In young poults the disease may be noticed as early as the fourth or fifth day following exposure. The poults appear listless and seek heat. There are watery droppings. The poults lose weight and become emaciated. Their temperature is usually below normal.

In growing turkeys the disease usually affects the flock suddenly. There is a sudden drop in feed and water consumption and the droppings are watery and brownish in color. The birds appear listless and the color of the head and skin appears dark and cyanotic. The birds lose weight rapidly.

Postmortem appearance is not too significant except for a spleen that is smaller than normal and the pancreas may be chalky white and show small pinpoint areas of degenerated tissue. The intestinal tract shows the greatest change. The contents of the small and large intestines are watery and there may be present mucous casts.

The diagnosis is based on autopsy findings and the history of the outbreak.

Recently antibiotics alone and in combination with other chemotherapeutic agents have been used to treat poultry flocks for avian monocyctosis and appear to be the drugs of choice. They will not completely prevent an outbreak.

MYCOSES

The mycoses or fungous diseases of fowls are of three sorts—aspergillosis, in which the air passages are involved, favus which affects the unfeathered portions of the head, and thrush, in which the mucous membranes of the upper digestive tract are affected. Each is the result of a different organism, but it is only recently that clear-cut and definite classification has been made possible through advances in systematic mycology.

Aspergillosis—This disease, which in chicks has often been called "brooder pneumonia" is the result of invasion of the air passages, especially the lungs and air sacs, by the fungus *Aspergillus fumigatus*. The mold and its spores are widely distributed and occur on many organic materials such as straw, grain, and the like. They are easily inhaled

by fowls when in contact with the moldy materials. Healthy, active fowls may often inhale the spores with no apparent ill effects, but when resistance is lowered through faulty nutrition, through close confinement in damp quarters, or in other ways, inhaling of the organisms may be followed by disastrous results. It is also probable that there is marked variation in individual resistance to the fungus.

The symptoms have been described by a number of writers. They usually appear in eight to twelve days after inoculation, according to Guberlet (1923), and consist of lessened activity, diminished appetite and slightly darkened comb, followed by droopiness, ruffled feathers, high body temperature, much darkened comb, complete lack of appetite and great thirst. As the disease progresses there is marked diarrhea, emaciation and weakness. In the advanced stages there is always a typical foul odor present. There is usually quickened breathing, sneezing and gasping for breath. After four or five days the fowls become prostrate, gradually pass into a comatose condition, and die.

At autopsy there will be found in the lungs, bronchial tubes and trachea masses of mycelia and spores in the form of white, gray or greenish nodules varying in size from that of a pinhead to that of a small pea. A positive diagnosis requires the identification of the fungus by microscopic examination of the filaments and spores taken from the lesions.

Guberlet (1923) reports a mortality ranging up to 50 per cent in certain flocks in Oklahoma. Savage and Isa (1933) describe a case in which the mortality in a flock of 400 young chicks exceeded 90 per cent. Dry corn ensilage from the bottom of a silo had been used as litter and it was found to carry the fungus.

Hudson (1947) reported seven outbreaks of *Aspergillus fumigatus* infection in the eyes of baby chicks, thus confirming similar observations by Reis in Brazil.

Moore (1953) reported several cases of aspergillosis in turkeys, and stated that it not only proves fatal to turkey toms but lowers the breeding efficiency of those which are less severely affected.

After the symptoms of aspergillosis have appeared, treatment is quite ineffective. Prevention appears to be simply a

matter of avoiding the use of any moldy litter or grain, and of keeping the fowls in good physical condition by the use of adequate rations and dry, clean, uncrowded quarters. Daily stirring of the litter in the brooder house, especially around and beneath the hover, makes conditions less favorable for growth of molds.

Favus.—This rather uncommon disease is a primary cutaneous mycosis characterized by yellowish-white, scaly lesions on the unfeathered parts of the head, particularly the comb. In severe cases it spreads to the neck and body, causing the feathers to become brittle and break off. Since it may be transmitted to man, care should be taken in handling affected fowls in order to avoid introducing the organism into cuts or scratches.

The cause of this disease is the fungus *Achorion gallinæ*. It may be spread from fowl to fowl by simple contact, and it is therefore important to isolate affected birds as soon as they are discovered.

Beach and Halpin (1918) were unable to produce the disease by feeding material taken from the combs of affected fowls, or by making intravenous injections, though it was easily transmitted by rubbing the material on the scarified combs of healthy fowls. These same workers report indifferent success in treating the disease by the usually recommended methods. They found, however, that it could be easily cured, except in severe cases, by one application of an ointment of formalin and petroleum jelly. To make this ointment, petroleum jelly should be placed in a jar which can be tightly sealed (such as a Mason fruit jar) and melted by setting the jar in hot water. When the petroleum jelly is melted add 5 per cent by weight of commercial formalin, tighten the cover immediately, and shake until the petroleum jelly hardens. One application of this ointment, rubbed thoroughly into the lesions, was sufficient to cure 50 out of 52 cases of favus.

Riedel (1950) was able to infect chickens with *Achorion gallinæ* only after scarification of the combs. Three weeks later the growth of the fungus was immediately arrested by the application of a 2 per cent solution of quaternary ammonium compounds consisting of equal parts of alkyl-dimethyl-

benzyl-ammonium chloride and alkyl-dimethyl-dichlor-benzyl-ammonium chloride. The fungicide showed no evidence of either dermal or ocular toxicity.

Thrush.—Although not often of great importance, thrush may assume serious proportions, as shown by Jungherr (1933). He reported a loss of 64 per cent among young chicks on one plant, and a total loss of 10,000 chicks among some 50,000 put out by a commercial hatchery. The disease has also



FIG 67.—An inverted crop of a chick affected with thrush or sour crop (moniliasis). The whitish round ulcers are characteristic. (Courtesy of Storrs Agricultural Experiment Station)

been recognized in turkeys, pigeons, pheasants, quail and grouse. It is characterized, according to Jungherr, by whitish ulcers of pseudomembranes in the crop, brownish or mucoid deposits in the proventriculus, and ulcers in the gizzard. The lesions in young chicks are often so small as to be easily overlooked.

The condition is primarily caused by the yeast-like fungus *Candida albicans*, and more rarely by *Oidium* species. Yeast-like fungi do not appear to be commonly associated with diseases other than thrush cases, and the etiological impor-

tance of the organisms has been shown by epidemiological pathological and cultural evidence. Jungherr was able to reproduce the disease by feeding fecal material from diseased chicks and by injecting fungus cultures of the *Candida albicans* type. The average period of incubation under experimental conditions was thirty-one days. The fungus was isolated from the crop, proventriculus, gizzard, gall bladder



FIG 68—Section through the crop of a chick affected with thrush or sour crop (moniliasis) showing root-like threads of *Candida albicans* invading the mucous membrane ($\times 200$) (Courtesy of Storrs Agricultural Experiment Station)

and intestine of affected chickens. Yeast like fungi were also recovered from the cloaca of laying hens affected with a moist type of "vent gleet."

According to Saunders (1948) *Candida* seems to form a part of the normal flora of the human and animal digestive tracts. Invasion of the tissue probably occurs only under the influence of some predisposing factor. For a reliable diagnosis actual tissue invasion should be demonstrated histologically, and in wet preparations from lesions. There

is little or no evidence to show that the disease is transmitted from animals to man

It is not known where nor how long the organisms can persist outside the animal body, nor just how the disease is normally transmitted. Jungherr has presented evidence which indicates that it may be transmitted through the agency of the egg, presumably on the shell.

Treatment of thrush is ineffective, so far as known, as the fungus is highly resistant to the common disinfectants.

TUMORS OF THE FOWL

Tumors (neoplasms) are of frequent occurrence in domestic fowls and, as in the higher animals, are of many different types. They affect various organs and structures of the body and may attain a relatively enormous size. In the past they have not been considered as important from an economic standpoint, but it now appears that either their incidence is on the increase or more care is being taken to report their occurrence.

Tumors are masses of new tissue that grow independently of the mechanisms which regulate the growth of the rest of the body. They are made up of the same varieties of cells that exist in the normal body. Many peculiarities are exhibited by neoplasms in their growth as, for example, the presence of a lipoma (fat tumor) in an extremely emaciated body devoid of any normal fat. Tumors are named from the type of cells of which they are comprised, not from the organ or part of the body in which they are located. The suffix "oma" is used to designate a tumorous condition, being appended to some word which indicates the type of tissue constituting the tumor, *e. g.*, osteoma, a bone tumor, lipoma, a fat tumor, or myoma, a muscle tumor.

There are two principal clinical types of neoplasms—benign and malignant. Benign tumors increase in size by central growth and are not invasive, whereas malignant tumors attack and replace the surrounding tissue. Benign growths do not spread from one organ to another, but may do irreparable damage by their growth and subsequent interference

with some of the vital body functions. Malignant neoplasms on the other hand, are prone to spread or metastasize and may thus be found involving several structures, ultimately destroying and replacing them.

The cause of tumors is not known, but several theories have been advanced to account for them. Among the hypotheses are (1) the misplacement and subsequent unrestrained growth of embryonal cells, and (2) the release of a group of cells from the growth restraint ordinarily imposed upon body tissues. Irritation is known to induce tumor formation at times, and the experimental injection of certain irritating substances is followed in many instances by the production of neoplasms.

Abnormal activity of tissue may also be productive of tumors. The frequency with which ovarian tumors occur in fowls is well known, and this has been ascribed to the exceptional state of activity to which the ovary has been brought by selective breeding for egg production. It has been shown in some instances that the cause of certain fowl tumors is a filterable agent, which upon injection regularly produces new growths. As a rule however, tumors are not transmissible. Age is apparently a factor in the incidence of neoplasms in fowls, for they are far more prevalent in birds over two years of age than in younger fowls.

A detailed description of tumors is not within the province of this discussion, and only brief mention will be made of a few of the types more commonly found in fowls. The reader who wishes to obtain full details regarding neoplasms will find abundant information in the list of references at the close of this chapter.

Sarcomas are malignant growths having their origin in connective (supporting) tissue. They are of frequent occurrence, and one type (Rous sarcoma No. 1) is capable of being regularly transmitted from bird to bird. Sarcoma may be found in practically every part of the body, especially in the skin, periosteum of bones, bone-marrow and cartilage, and less frequently in the respiratory, reproductive and urinary organs.

Tumors of this type frequently combine with other neo-

plasms to produce a mixed tumor. If, for example, a sarcoma is associated with a tumor having a glandular structure, the resulting combination would be termed an adenosarcoma, if with a fibrous-tissue tumor, the combination would be a fibrosarcoma, and so on. Adenosarcomas, lymphosarcomas and myxosarcomas have all been reported as occurring in fowls. *Other connective-tissue tumors include the lipoma (fatty tumor), the myxoma (mucous tissue tumor), the chondroma (cartilage tumor), and osteoma (bone tissue tumor).* Many other types and combinations exist in the connective tissue group, but most of them are of rare occurrence in fowls.

Carcinomas are malignant tumors of epithelial origin, and do not occur so frequently in fowls as do the sarcomas. They often combine with other types of tumors. Squamous epitheliomas are malignant carcinomas occurring in the skin and membranes lined with squamous epithelium. Epitheliomas have been reported as occurring in the kidneys and ovaries of fowls.

In a study of the frequency of occurrence of tumors in fowls, Feldman (1927) found leiomyomas, lymphocytomas, lymphosarcomas, cystadenomas, adenocarcinomas and carcinomas. Mathews and Walkey (1929) state that lymphoid tumors are of frequent occurrence in fowls, and describe in detail their findings in a series of 168 cases of such neoplasms. The growths were found in birds ranging in age from six weeks to three years. In one outbreak the mortality was 30 per cent during the first three months. *The symptoms were listlessness, capricious appetite, gradual emaciation, roughened feathers, diarrhea in the final stages, pendent abdomen in some cases, sunken eyeballs and pallor.* The postmortem changes were subcutaneous edema along the neck and sternum, with diffuse or nodular tumor formation. The tumors were found in the ovary, liver, kidney, spleen, pancreas, proventriculus, gizzard, intestine, heart, lungs, muscles, testicles and thyroids. Despite the fact that this series of tumors was purely lymphoid in nature, the authors state that they should not be confused with the tumorous swellings sometimes observed in "fowl paralysis."

REPRODUCTIVE DISORDERS

Various troubles associated with the reproductive organs of the hen are common in poultry flocks and, taken together, cause a considerable loss. Many of them are not amenable to treatment, but because of their importance they are worthy of some consideration.

Abnormal Eggs—Many sorts of abnormal eggs are produced by fowls at different times. Soft-shell or shell-less eggs may result from an insufficient supply of shell-forming material in the diet, from premature laying before the shell was completely formed, or from a failure of the uterus or "shell gland" to function properly. Correction of the condition has sometimes been obtained by the feeding of calcium gluconate, this salt apparently having the property of exerting an immediate influence on shell formation.

Double-yolked eggs are quite common. They are laid by a rather small percentage of the females in a flock, and are more likely to be laid by pullets than by hens. Triple-yolked eggs are rare. Either kind results from more than one yolk being in the oviduct at a given time.

Eggs are sometimes laid with more than one shell covering, with rough wrinkled, or otherwise abnormal shells, or with foreign bodies such as blood clots within the shell. So far as is known there is no cure nor any preventive for these conditions. Such eggs can be detected readily by candling.

Cage Layer Fatigue—This name has been given to a condition which occurs in pullets kept in individual cages, and described as a type of leg trouble which disappears when the affected birds are removed from the cages. Francis (1957) reported significant differences between strains in the occurrence of the disorder. It has been suggested that a nutritional deficiency of some kind is responsible but no specific cause has yet been demonstrated.

Egg-bound—Poultrymen describe a hen as being "egg bound" when it is obvious that she is trying to lay an egg and cannot do so. The condition is similar to that of difficult parturition in mammals. It may result from inflammation of the oviduct, partial paralysis of the muscles of the oviduct, or the production of an egg so large that it is physically

impossible for the hen to lay it. Less frequently, it may result from actual obstruction of the oviduct by a stricture, or by twisting of the duct in such a way as to close it, or by the accumulation of exudate following severe inflammation.

Many cases of egg-bound recover naturally without treatment. It is often possible to insert a finger, well lubricated with carbolized vaseline, into the cloaca, and by manipulation with the other hand to expel the egg. If this is not successful, the egg may be brought to a position such that the end of the shell is externally visible and held there while the shell is broken and the contents and pieces of shell removed. Injections of cold water two or three times daily for a few days will be helpful. The fowl should be kept as quiet as possible until it appears to be normal again.

Eversion of the Oviduct—This condition, which is often referred to as prolapse or "blow out," is commonly the direct result of straining on the part of the bird in an effort to lay. It often follows the condition of egg-bound, which has just been described. The oviduct is highly congested and with the cloaca, is everted through the vent. If not discovered promptly, it may easily be the immediate cause of an outbreak of cannibalism in the flock, because other birds in the flock will pick at the extruded mass until the victim is killed.

It is also possible for picking to be the inciting cause of prolapse. Females which have been picked inside the vent may, because of the ensuing inflammation and irritation, continue to strain in an effort to dislodge the irritating material and eventually evert the cloaca in a case of prolapse. Scott (1948) has observed numerous cases of such picked individuals in a flock where a few cases of prolapse have occurred.

Stafseth, Thompson and Grey (1932) made rather extensive observations of the occurrence of this trouble in Michigan flocks. No specific cause was found, but the authors incline to the opinion that "prolapse is a symptom of certain diseased conditions in the intestine and more rarely in the oviduct. Hens in their first year of production, and especially White Leghorns, are the chief sufferers. We agree with others that prolapse of the rectum and oviduct and eversion of the cloaca are usually associated." These authors

further state that "as a cause of the intestinal inflammation so frequently found in these birds, we feel that worms and coccidia play a very important part, and the reason for less prolapse occurring in older birds than in young ones may lie in acquired immunity and not merely in physical strength acquired with age"

Burmester (1948) found that White Leghorn females implanted with pellets of the male sex hormone had a significantly higher incidence of prolapse than did their sisters implanted with female hormone. He suggests that certain individuals have a tendency to prolapse either because of insufficient estrogens or because of excessive amounts of androgens. The effect is presumably brought about by hormonal influence on ligaments of the oviduct, spread of the pubic bones, size of the cloaca and vent, and pliability of the abdomen.

Wheeler and Hoffmann (1948), on the other hand, reported four cases of prolapse in young cockerels following implantation with 15-mg pellets of diethylstilbestrol at twelve weeks of age. They, too, feel that prolapse and pick-out are in some way associated with the estrogen level of the blood rather than with straining, constipation, intestinal irritation, and the like.

If prolapse is discovered in time, treatment is often successful. The prolapsed tissue should be carefully washed with warm water and gently put back in place. The fowl should be isolated and kept as quiet as possible for a few days, and it is well to inject cold water or to insert a lump of ice into the cloaca two or three times daily in order to relieve the congestion and to promote contraction of the parts. If the condition recurs in the same bird, it is rarely worth while to give another treatment, the best procedure being to dispose of the fowl.

Rupture of the Oviduct.—It sometimes happens that in cases of complicated obstruction of the oviduct, the walls of the organ rupture and permit the contents to escape into the abdominal cavity. The trouble is not common, and usually is not found until a postmortem examination is being made of the affected fowl. Pearl and Curtis (1914) were able to show that rather extensive ruptures of the upper part of the

oviduct, if uncomplicated by obstructions in the duct, will heal spontaneously. They removed large pieces of the oviduct wall in experimental subjects and were unable, at later autopsy, to find any evidence of injury other than a small scar.

Finne and Vike (1951) described a type of hereditary atresia of the oviduct, observed in a flock of White Leghorns, which they designated as *atresia isthmi*. Females developed normally but were unable to lay because the oviduct was ruptured at the isthmus. The ovary was normal so that growth and ovulation of yolks proceeded normally. Yolks never reached the uterus, but passed instead into the body cavity where they caused peritonitis and death. The females usually died at five to six months of age shortly after egg laying would begin in normal pullets. In affected males there was clear rupture of the right seminal duct.

Salpingitis—This is a term used to designate an inflamed condition of the oviduct, in which no specific causative organism has been found. There is a discharge from the inflamed duct which causes irritation of the vent and gives a "smeary" appearance to the feathers just below it. It is a condition which predisposes the fowl to egg-bound, prolapse and cannibalism by other birds.

Treatment of salpingitis consists of the isolation of affected fowls and washing of the cloaca and lower portion of the oviduct with warm salt water (2 level teaspoonfuls of salt to 1 quart of warm water). This is best done by using a hard rubber syringe with a blunt nose and gently irrigating the involved organs. Since the trouble has been reported as sometimes traceable to infection with *Salmonella pullorum*, it would be unwise to use for breeding purposes any affected fowls which may recover.

Cloacitis—This is an ulcerative condition of the cloaca and vent, accompanied by an offensive odor, and is often referred to as "vent gleet". It is usually described as a venereal disease of fowls, but the causative organism has not been isolated.

The work of Goldberg and Benson (1920-1921), Gwatkin (1925), and Scherago (1925) indicates that the disease cannot properly be regarded as infectious or contagious, at least

Carver (1932) states that it can be quickly and effectively controlled by either one of two methods. If the chicks are being raised indoors where electric current is available, he recommends the use of 60-watt or 100-watt natural colored ruby Mazda bulbs, glass not inside frosted, to light the brooder room. All other light should be excluded. Reflectors to concentrate the light on the feeding and watering utensils are recommended. With colony reared flocks, or when electricity is not available, it is recommended that the inside of the windows be sprayed with rich, red lacquer. For a paint that is easily washed off at the end of the brooding season, it is suggested that dark red, opaque, flat-finish, show-card color be used.

From a practical standpoint it is much more important to provide brooding conditions which will make it unlikely that cannibalism will ever get started. An important point in this connection is the maintenance of a moderate, even temperature. In addition the chicks should have plenty of room, indoors as well as out, with ample opportunity to keep busy at harmless activities.

There has been a prevalent feeling among practical poultrymen that free-choice feeding of oats was helpful in preventing or controlling cannibalism. From a four-year study of this problem Miller and Bearse (1937) concluded that oats fed as the sole cereal in a ration for growing and laying pullets consistently and significantly reduced cannibalism below that experienced with corn as the sole cereal. Some difficulty resulted from impacted gizzards and pendulous crops in the groups fed the high levels of oats. It was also true that the oat fed groups were lowest in feed-efficiency. The results suggest, however, that there may be some deficiency in high-corn rations which is a contributing cause of cannibalism.

The results obtained by Scott and co-workers (1946) on rations high in corn but adequately supplemented with the nutrients in which corn is lacking or deficient suggest that a deficiency of tryptophane or of nicotinic acid or both may be responsible for many cases of cannibalism. Schuble, Davidson and Binderer (1947) observed that malnutrition may of itself result in feather picking and cannibalism in Leghorn

chickens They found that quality of feathering was improved by the addition to a high corn ration of those ingredients which reduced feather picking and cannibalism

According to tests made at the Beltsville Research Center and at the University of Wisconsin, cannibalism can nearly always be checked by the salt treatment This consists of increasing the salt content of the mash to about 4 per cent mixture, if both grain and mash are being fed, or to 2 per cent if an all mash ration is used Feather picking or cannibalism will usually stop promptly, but in some cases the high salt feeding may have to be continued for three or four days It is important to use the high salt feeding for only a short time, though it may be repeated at frequent intervals, if necessary Continuous salt feeding is much less effective An alternative method of giving the salt is to add it to the drinking water, for a half-day at a time at the rate of 1 to 2 tablespoons to each gallon of water

EDEMA OF THE WATTLES

This is a condition in which the tissue of the wattle is filled with inflammatory fluid so that the appendage becomes greatly enlarged In most cases only one wattle is affected It is swollen and hot, red at first, turning to yellowish gray as the disease progresses In severe cases the swelling may involve the head The liquid often becomes caseated into hard, cheesy nodules, which may be removed without difficulty Males are more commonly affected than females The disease usually runs a chronic course, and the death loss is not high In recovered fowls the wattle which was diseased has a shrunken and wrinkled appearance

The cause is not definitely known, but the organism of fowl cholera *Pasteurella multocida* has frequently been isolated from affected fowls and has been suggested as the probable cause of the trouble Bushnell and Twiehaus (1945) state that the infection usually gains entrance through wounds in the wattle caused by fighting

Delaplane and Higgins (1948) believe that in some parts of the country the respiratory form of fowl cholera, characterized by nasal discharge, swollen and distended sinuses,

and swollen wattles, is the most important respiratory disease of bacterial origin. Their studies indicate that sulfa-quinoxaline used continuously at the rate of 0.033 per cent in the mash is of value in preventing the spread of the disease.

FROZEN COMBS AND WATTLES

In the northern parts of the country it is not unusual for the combs and wattles, especially of male birds, to become badly frozen during extremely cold weather. Freezing of the wattles is especially common because they are so frequently dipped into the drinking water. The frosted parts are often swollen and extremely painful. They turn bluish-red in color, and the severely frozen portions slough off.

If discovered early, the affected parts may be thawed out with snow or cold water, after which they should be well greased with petroleum jelly. If severely frozen birds are not discovered until the parts are badly swollen, it is often best to amputate the comb or wattles, as the case may be, in order to facilitate a prompt recovery. The danger of freezing can be largely eliminated by early dubbing of the comb and wattles, if desired.

NECROSIS OF THE BEAK

Conklin and Maw (1930) have described a condition in which necrosis of the beak followed the feeding of a very finely ground all-mash ration to chicks in battery brooders. Some of the finely ground feed remained under the tongue and adhered to the outer edges of the upper and lower mandibles, setting up an inflammation of the tissues and permitting secondary infection. In severe cases there was ultimate loss of the lower mandible, and in a few cases the upper mandible as well. The impaction commenced in chicks about four weeks of age. The condition was easily cured by removing the adhering food material, disinfecting the mouth parts to avoid further infection, and changing the ration to one of more granular consistency. Ground wheat or wheat flour middlings are likely to be especially troublesome because of their gluten content.

PENDULOUS CROP

Enlarged, pendulous crops are not uncommon, especially among the heavier breeds of fowls. The loose, baggy condition of the empty crop seems to cause little inconvenience to the affected fowl, and treatment is not often worth while. It is probable that the condition can be greatly relieved, and perhaps entirely cured, by surgical removal of a piece of the crop-wall. The same general procedure as described for the relief of impacted crop should be followed in performing the operation.



FIG 69 —Necrosis of the beak (Courtesy of W A Maw)

Hinshaw and Asmundson (1936) reported observations on occurrence of this trouble in turkeys, the yearly incidence being 10 per cent of one flock in 1934. In a group of 206 affected turkeys, 35 per cent recovered, 35 per cent died as a direct result of the condition, 4 per cent died from miscellaneous causes and the remainder were killed either because of emaciation or because they were unfit for market.

Most of the cases occurred when the poults were from nine to twelve weeks of age, and were associated with excessive liquid consumption during heat waves that were accompanied by extremely low humidity. The obvious conclusion is that careful management may help prevent the trouble. The

authors also suggest that selection of resistant breeding stock appears to be a logical means of prevention

HEAT PROSTRATION

Fowls are adversely affected by excessively high temperatures, especially when accompanied by high humidity, and heat prostrations are not of unusual occurrence. Prompt removal to a cool place and cooling of the body by temporary immersion in cool water may bring about recovery of stricken birds, but death often follows in spite of anything the caretaker may do. Provision of shade, of ventilated nests and of an ample supply of water at all times, are helpful preventive measures. Frequent trips to release trapped fowls on hot days are especially important. In feeding stations where large numbers of chickens are kept in fattening batteries, it is common practice to spray water directly on the birds two or three times daily during extremely hot weather.

Stiles (1943) reported a case in which there were many victims of heat exhaustion in a flock of 600 poults which were abruptly transferred at three weeks of age from relatively cool battery brooders to quarters which were extremely hot (105° F and above). The affected poults showed low body temperature, sweating, prostration and death. Recovery of some poults was brought about by application of heat. Shade and ventilation corrected the trouble.

Lee *et al* (1945) reported extensive observations on the reactions of fowls to hot atmospheres, and on practical methods for relieving their distress so as to avoid excessive mortality and extreme losses in egg production. They emphasize the importance of shade, adequate ventilation, and an ample supply of drinking water. Wilson (1948) observed that White Leghorn pullets exposed to an ambient temperature of 95° F consumed twice as much water as when kept at a temperature of 70° F.

KEEL CYSTS

Chickens and turkeys of market size often develop cyst-like formations between the skin of the breast and the keel

bone. These growths are commonly referred to as breast blisters, a term which is technically incorrect inasmuch as a blister is a vesicle in the skin. They are most common in males of the heavy breeds and are rarely seen in females or in White Leghorns of either sex.

Keel cysts for which O'Neil (1943) suggests the scientific name *bursa synorialis presternalis* vary in size from that of a small bean to a formation nearly three inches in length and $\frac{3}{4}$ inch in diameter. They usually contain a cavity which is filled with a brownish sterile viscous fluid. No specific cause is known other than mechanical pressure and friction. Bird (1944) found the incidence and severity to be dependent upon depth of the fowls through the pectoral region relative to body weight. He further found by the use of an ingenious device for measuring keel pressure that weight or pressure against the roost during perching is highly correlated with relative body depth. Presumably selection for broad breasts would be helpful in reducing the incidence of keel cysts.

TOXIC HEART DEGENERATION

Fischel (1946) has described a disease of unknown etiology occurring in New Zealand and characterized by an enlarged and parboiled appearance of the heart. Blaxland and Markson (1947) state that a typical round heart as found in this disease is about one-fourth heavier than normal because of a thickened myocardium. The apex of the heart is markedly rounded. Bacteriological examinations and transmission experiments have been completely negative. No method of treatment is known. The only clue to the possible nature of the disease is that lesions found in the heart muscles are similar to those described by Jungherr and Levine (1941) as occurring in pullet disease (avian monocytosis).

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methods of detoxication, but they are of interest primarily to the student of toxicology.

DRUGS AND CHEMICALS

Most of the drugs and chemicals which are likely to cause illness or death of poultry belong in the class of so-called mineral poisons. Fowls ordinarily have access to them only as the result of accident or carelessness, but it is helpful to have some idea of their relative toxicity.

Ammonium Chloride.—This drug would not often be used in poultry practice. It has been suggested in the treatment of ascites (abdominal dropsy), but surgical relief is usually preferred. The non-toxic dose is reported by Gallagher(1919) as 15 to 45 grains for a mature fowl. The toxic and lethal doses are given as 60 grains in dry form or 45 grains in solution.

Arsenic.—The danger of poisoning fowls with rat poisons, Paris green, lead arsenate, or other arsenicals should not be overlooked, though if reasonable precautions are taken in the use of such materials the likelihood of poisoning fowls is rather remote.

It has been shown by Barber and Hubster (1933) that affected birds may develop a tolerance to arsenic, after which they will be fatally poisoned only by heavy doses. Such relative immunity is established only through the administration of small daily doses over a long period. These workers state that the lethal dose of arsenious acid ranges upward from 4 grains at a single ingestion, a value which is in substantial agreement with the minimum lethal dose of 5 grains given by Gallagher.

The question of arsenic poisoning becomes important under conditions such as exist in South Africa where it is common practice to use as a protein supplement in poultry rations a locust meal made from poisoned locusts. Van Zyl (1929) reported the minimum lethal dose of ordinary white arsenic (arsenious oxide) to be about 0.075 gram of As_2O_3 per kilogram of body weight, or about 150 mg. (2½ grains) per head for 4½-pound fowls. This probably would vary among individual fowls and with the fineness of the arsenic. He found

Chapter 11

POISONS

CASES of poisoning are not very common when considered in relation to the many other ailments of poultry, but it is nevertheless true that fowls frequently have access to poisonous substances, and that under certain conditions they are readily subject to either acute or chronic poisoning. Under normal range conditions, with plenty of green forage available, the sense of taste protects fowls from many poisonous materials, but when they are closely confined in bare yards, or when the supply of natural food is short as a result of prolonged dry weather, they are quick to eat almost any succulent material offered, even though it may be of a poisonous nature.

In the great majority of cases, by the time an accurate diagnosis of poisoning has been made it is too late for treatment to be of any help to the affected fowls. The reason for discussing the various poisons in some detail here is to aid the flock owner and practitioner in locating the source of poisoning as promptly as possible so that it can be removed for the protection of the remaining fowls.

A study of what happens in the body of the fowl after the ingestion of poisonous materials has been made by Sherwin and Crowdle (1922), and a brief review of their findings may be of interest. The first action of the animal body in its efforts to deal with toxic substances is an attempt at complete oxidation of the foreign molecule. Should this fail, an effort is next made to render the compound less toxic by means of reduction, or the removal of oxygen. If neither of these types of reaction sufficiently alters the foreign material, an attempt is finally made to combine it with some other compound so as to form a new substance which is less toxic. The general effect is to produce alcohols or acids which the body can eliminate. Numerous differences have been found among fowls, humans, dogs and rabbits in respect to exact

methods of detoxication, but they are of interest primarily to the student of toxicology.

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the toxicity of soluble sodium arsenite to be just twice that of the white arsenic

In Van Zyl's experiments, fowls were able to tolerate considerable quantities of arsenic over an extended period, and meal prepared from locusts showing a high arsenic content was quite harmless when fed in as large quantities as the fowls would consume. He concluded that the danger of poisoning poultry from the feeding of locust meal made from poisoned locusts is practically negligible.

Whitehead (1934) has reported the results of an extensive study of the danger to chickens through the use of poisoned bran in grasshopper control. By forced feeding of poisoned bran it was found that 74 mg ($1\frac{1}{4}$ grains) of white arsenic constituted a slightly toxic dose for a 22-ounce chicken. This is at the rate of 119 mg per kilogram of live weight.

It was also observed that chickens readily recognize poisoned grasshoppers and will eat less than one-half as many of them as they will of unpoisoned grasshoppers. Even though no other food was available chickens did not eat enough poisoned grasshoppers to obtain a toxic dose of arsenic.

The arsenic obtained in this way did not have a cumulative effect, even though the fowls were fed for a period of sixty-six days. Feeding on poisoned grasshoppers did not materially affect the weight or growth of chickens.

Chickens never consumed enough arsenic at one time to constitute a dangerous dose for humans, and chemical analyses of the bodies of chickens showed definitely that there is no danger of secondary human poisoning from arsenic storage in the flesh of chickens that have eaten poisoned grasshoppers.

Other workers, including Chorley and McChlery (1935) and Wilson and Holmes (1936), have obtained similar results under experimental conditions. In spite of such evidence, occasional field cases are reported in which there is apparent chronic or acute arsenic poisoning either from poisoned bait or from poisoned grasshoppers. Serles (1937) is of the opinion that chronic poisoning presents the major problem, and that it is more frequent than is commonly supposed.

Thomas and Shealy (1932) reported that though lead arsenate will produce death in chickens when fed in large

quantities, spray solution containing 4 pounds of lead arsenate in each 100 gallons was not harmful when consumed with feed and water continually for sixty days. As much as 13 grains of lead arsenate per bird daily for sixty days did not cause any apparent ill effects on chickens of 2 to 3 pounds weight. Such drip as normally occurs from fruit trees following the use of arsenical sprays is probably not a source of danger.

Hundreds of arsenical compounds have been tested for their ability to control parasitic diseases. When arsanilic acid, which is the primary intermediate in the manufacture of many other arsenicals, was shown to have growth stimulating properties for chickens and turkeys it became necessary to determine its possible toxicity at feeding levels. Frost (1953) in reviewing the pertinent literature stated that (1) poultry show a high degree of tolerance to arsanilic acid, (2) arsenic retention is greatest in the liver, least in muscle, and is proportional to the level fed, and (3) arsenic retained by chickens does not appear to be a health hazard to consumers of poultry meat.

Barium Carbonate—In a field case of barium carbonate poisoning reported by Newton (1941), there were 185 deaths in a flock of 300 fowls. Symptoms included darkening of the comb, distressed appearance, and incoordination of gait. Lesions included congestion of the alimentary tract, shedding of the lining membranes of the crop and stomach, and congestion of the liver. The lethal dose for a 1600-gram chicken was determined to be between 6 and 10 grains.

Boric Acid—*Canned string beans prepared with a commercial canning compound which consisted chiefly of boric acid, and used at a rate which resulted in 9 grams of boric acid per quart of canned beans, were found to be poisonous to young chicks and mature hens (Gallagher, 1924). The fowls would not eat such preserved food after the first experience. Forced feeding resulted in extensive inflammation and necrosis of the crop, proventriculus, and intestine, followed by death.*

Copper Sulphate—This material, also known as blue vitriol, has sometimes been recommended for medicating the drinking water given to fowls. That it is distinctly poisonous is shown by the work of Gallagher (1919) who found that 20

grains of the dry material or 15 grains in solution constituted a fatal dose

Cyanides — According to Gallagher (1919) fowls are less resistant than dogs to potassium cyanide 1 to 2 grains amounting to a lethal dose and as little as $\frac{1}{16}$ grain being toxic. He reported further that absorption of such substances as ammonium chloride and potassium cyanide through the crop wall is very rapid. Symptoms appeared in from two to five minutes after the administration of these poisons.

The extreme toxicity of calcium cyanide for fowls is evident from the report of Winchell (1925) who used it in both the dust and flake forms for the destruction of some 25 000 or 30 000 birds during the course of control work with European fowl pest. The fowls were in live-poultry cars, shipping crates and covered trucks. Using 1 pound of calcium cyanide for about 2000 fowls the dust was blown on the fowls from the windward side or the flake was sprinkled on the floor. Fowls died within one or two minutes almost without struggling.

It should be obvious that extreme caution must be used when dealing with any of the cyanides. Hydrogen cyanide for example is sometimes used to fumigate a straw loft in order to destroy rats. Care should be taken to see that the entire building is thoroughly ventilated before allowing either fowls or persons to enter.

Insecticides — With the introduction and widespread use of several new insecticides it is important to know whether there are any direct or indirect toxic effects on poultry.

Alrin, a naphthalene compound effective against the fowl tick, is highly toxic to poultry as well as to humans and must be used with care. Skin contact, inhalation and food contamination should be avoided. Tests carried out by Andersen, Blakely and McGregor (1952) showed that all ingested levels above 2 ppm depressed growth in young turkeys. Levels above 25 ppm were highly toxic. Dieldrin, another naphthalene compound, would presumably have similar effects.

Chlordane, a mixture of several chlorinated compounds when used as a 2 per cent insecticide spray was found to be

highly toxic to turkey poults for as long as one week after its use (Moore and Carter, 1954)

DDT (dichloro diphenyl-trichlorethane), widely used for fly control and in the control of insects infesting stored grain, is definitely toxic if consumed directly at levels above about 0.03 per cent. There is no appreciable danger of indirect poisoning of young or old fowls from the feeding of grain treated with DDT or from the ingestion of flies killed by DDT sprays.

March and co workers (1956) studied the fate of malathion in the laying hen by labeling it with radio-active phosphorus. When they fed 100 ppm in the mash they saw no toxic effects and found that 75 per cent of the compound was eliminated within five or six days mostly as water-soluble metabolites and degradation products. Maximum residues found in tissues or eggs were less than 3 per cent of the concentration fed.

Kamala—This drug, which is often used as a remedy against tapeworms, acts as an irritant in the gastro-intestinal tract and also has a strong purgative action. To that extent it must be regarded as a poison. Maw (1934) has reported that the administration of 1 gram (15 grains) of kamala each to pullets shortly after they started to lay, resulted in a decline in egg production and a decrease in egg weight. Each of the separate parts of the egg decreased in weight, the decrease being greatest for the yolk. It was concluded that from a practical standpoint, a decline in egg production and a decrease in egg weight are to be expected when kamala is given to a laying flock.

Lead—Lead poisoning of domestic fowls is probably most often brought about by eating paint skins but in the case of wild waterfowl it frequently results from the ingestion of lead shot which accumulate in the mud of the shooting grounds. The symptoms which have been observed include paralysis of important muscles, which increases steadily as the ailment progresses, drooping of the wings, inability to walk and later to stand, thin, watery, greenish feces (the color being a well-marked symptom), a rapid, strong pulse, spasms in captive wild birds, and an effect on the heart of such a nature that a bird may die suddenly after fright or exertion.

metallic lead and were found to be variably and moderately toxic. Metallic lead was more toxic than these other heavy metals, and the mortality following its use was higher.

Mercury — When using mercurial ointment in the treatment of fowls for lice it is well to make certain that no excess of the ointment is left on the feathers where it can be picked off and eaten by the fowls. Glover (1932) has reported that excessive applications of mercurial ointment (containing metallic mercury) resulted in emaciation, diarrhea, dullness and paralysis. Postmortem examination revealed petechial hemorrhages in the duodenum and abdominal cavity, marked inflammation of the ceca and pronounced ecchymosis of the heart and liver. It is assumed that the poisoning followed ingestion of the excess ointment by the fowls. As Pearl, Surface and Curtis (1915) have remarked, a very large amount of the ointment would have to be applied to bring about mercurial poisoning through absorption.

Mercuric chloride (HgCl_2) is extremely poisonous to baby chicks. Parker (1929) found that 0.2 cc. of a 2 per cent solution proved fatal to nearly 40 per cent of the chicks to which it was given. The lethal dose was reported by Gallagher (1919) as 4 grains for a 4-pound fowl. Three grains was a non-toxic dose.

Fowls suffered no apparent ill effects during an eighteen-day period in which the only water they had to drink was a 1 to 6000 solution of mercuric chloride. When thirsty fowls were given 1 to 2000 and 1 to 4000 solutions they tasted them and refused to drink. Similar results with poults were reported by McNeil and Hinshaw (1945). They could find no evidence to justify such use of the drug in disease control.

Naphthalene — The ingestion of naphthalene moth balls, which had been placed in the nests as protection against lice and mites, resulted fatally according to Hudson (1936). Forty hens in a flock of 400 were lost with identical symptoms, including greenish-black diarrhea and paralysis of the legs. On autopsy, small blister-like areas were found in the mucosa of the crop. The liver was swollen and showed small necrotic areas. The intestines were catarrhal, and the gizzard contents gave off a strong odor of naphthalene. Removal of the moth balls from the nests ended the trouble.

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Nicotine Sulphate — This material is known to be extremely poisonous, though the toxic and lethal doses for mature fowls seem not to have been reported. Parker (1929) working with baby chicks, found that in 0.2 cc doses an 8 per cent solution killed every chick to which it was given, a 6 per cent solution killed 70 per cent of the chicks, and a 4 per cent solution killed 50 per cent of the chicks. With a 3 per cent solution there was an almost immediate reaction, practically identical for every chick, in the form of a complete coma lasting for about fifteen minutes, but there were very few deaths.

Phosphorus — Poisoning may result from the ingestion of rat poisons which contain phosphorus, though it would appear that rather large doses are necessary to produce sudden death. It has been pointed out by Clough (1930) that in birds "live" phosphorus remains unoxidized for a considerable time. Beaudette, Hudson and Weber (1933) report that it can usually be detected in the crop and gizzard by its characteristic odor of 'wet matches'.

Potassium Nitrate (Saltpeter) — A case of accidental poisoning in a flock of poultry fed potassium nitrate instead of the intended Epsom salts was reported by Guberlet (1922). Experiments conducted later indicated that the lethal dose is from 60 to 70 grains for $3\frac{1}{2}$ - to $4\frac{1}{2}$ -pound hens. Smaller doses cause diuresis and diarrhea. The toxic effect is lessened by keeping the system flushed with water. The symptoms observed included gastro-enteritis, muscular weakness, slow weak pulse, darkness of the skin, discolored comb, followed by subnormal temperature, paralysis, collapse, coma and death.

Potassium Permanganate — This material which has often been recommended as an antiseptic to be added to the drinking water, is decidedly poisonous if consumed in any appreciable quantity. Gallagher reported 15 grains as a non-toxic dose but found 30-grain doses to be lethal to adult fowls. Mature birds were not adversely affected by being forced to use as drinking water a 1 to 500 solution of potassium permanganate.

The statement has sometimes been made that the use of potassium permanganate in the drinking water when sour

milk is being fed results in the formation of a poisonous substance which is fatal to chickens. Eriksen (1924) fed young chicks all the sour milk they would consume and all the medicated water they could drink, using $\frac{1}{4}$ teaspoonful of potassium permanganate to each gallon of water, and observed no ill effects. In experiments with mature hens, sour milk and 0.2 per cent permanganate solution were introduced directly into the crop, separately and mixed together, and no ill effects resulted. He concluded that there is no need to discontinue the feeding of sour milk when, for any reason, it is desired to add potassium permanganate to the drinking water.

Sodium Bicarbonate — The use of sodium bicarbonate as a mild laxative for growing chicks has been rather common among poultry men in certain areas, but it has been shown by Witter (1936) that this is a questionable practice. He states that young chickens were injured by the material when given in 0.6 per cent solution, and that deaths occurred in chicks under eight weeks of age when given double this dosage. Mature cockerels were injured by a 2.4 per cent solution, but did not seem to be affected by a 1.2 per cent solution.

Affected chicks showed an increase in kidney weight, and these organs became pale, swollen, and engorged with urates. From this report, and from the work of Delaplane (1934) and Jungherr (1935), it appears that large single doses or repeated small doses of baking soda may cause lesions which are indistinguishable from those found in natural internal gout.

Sodium Chloride (Common Salt) — That an excess of salt in the feed is toxic to fowls, and may easily result in death, has been well established, though experimental feeding of salt has shown that the salt toleration of both chicks and older fowls is much higher than has sometimes been reported. The minimum lethal single dose for baby chicks and for mature fowls has been determined as close to 4 grams per kilogram of body weight, or 0.4 per cent of the live weight. Mitchell, Card and Carman (1926) found, however, that chickens could be raised from nine to twenty-one weeks of age on rations containing as high as 8 per cent of salt with no apparent detrimental effects. Quigley and Waite (1932) showed

that young chicks were able to endure salt levels as high as 30 per cent for short periods of time, and that it was impossible to place enough salt in an all-mash ration to produce an appreciable amount of sudden mortality. The unpalatability of feeds or rations containing dangerous levels of salt, and the tendency to consume large quantities of water when fed a salty ration, appear to act as a protection against an overdose under natural conditions.

Barlow, Slinger and Zimmer (1948) found 3 per cent added salt to be the minimum toxic level in chicks under nine weeks of age. The heaviest mortality in their experiments occurred during the first month after hatching.

Doll, Hull and Insko (1946) reported that water from a "soda-water" well containing chlorine equivalent to 0.54 per cent NaCl and sodium equivalent to 0.7 per cent NaCl was toxic for young chicks. Experimental use of sodium chloride in 0.5 per cent and 0.9 per cent concentration as drinking water produced disease in day-old chicks which was identical with that resulting from the well water. No toxicity resulted from the use of drinking water containing 0.25 per cent sodium chloride.

It is clear from this and other experiments that a given level of salt is much more toxic in the drinking water than in the feed. Scrivner (1946) found that comparatively low levels of sodium chloride or sodium bicarbonate produced edema and ascites in starting poults. Matterson, Scott and Jungherr (1946) observed edema in young poults fed 2 and 4 per cent of potassium chloride, and suggest that the lack of complete agreement on the sodium chloride tolerance may be due to differences in composition of the rations used, especially in respect to salts other than sodium chloride.

Sodium Fluoroacetate—This rodenticide, commonly known as "1080", is less toxic for chickens than for small mammals. The maximum tolerant dose has been determined by Cottral, Dibble and Winton (1947) to be from 4 to 5 mg. per kilogram of body weight, and the lethal dose from 15 to 18 mg.

Symptoms of "1080" poisoning were cyanosis of the comb and wattles, depression, weakness, disinclination to eat or move about, distention of the crop with liquid and gas, edema of the wattles, dyspnea, and moist rales.

Necropsy findings included hemorrhage and edema of the lungs, petechial hemorrhage in the pericardial and mesenteric fat, enteritis, congestion of the internal organs and, in laying fowls, ovarian hemorrhages. Hemorrhage and edema of the lungs are considered to be diagnostic.

Strychnine.—It is generally conceded that fowls are more resistant to strychnine than are mammals, but actual data on the lethal dose for fowls are very few. Pierce and Clegg (1915) reported the minimum lethal dose for quail by subcutaneous injection as 0.04 gram per kilogram of body weight, and Gallagher reported the lethal dose for chickens, when fed the poison, as equivalent to 0.08 gram per kilogram of body weight. This difference is substantiated by the work of Heinekamp (1925), who found that pigeons and chickens possess a relative immunity to strychnine only when the dose is given by mouth. The minimum lethal dose was found to depend on the contents of the crop, the rate of absorption being inversely proportional to the amount of food in the crop and directly proportional to its fluidity.

Sulfonamides.—The demonstrated bacteriostatic and coccidiostatic effects of various sulfa drugs have led to wide experimental study of their value in poultry disease control. Certain of these drugs, in particular sulfanilamide, have definite toxic effects on chickens and therefore have limited therapeutic value in practice. Levine (1939) found that as little as 0.1 per cent of sulfanilamide in the feed interfered with normal growth in young chickens. Scott, Jungherr and Matterson (1944) showed that concentrations of 0.25 per cent or more of sulfanilamide will inhibit egg production, and that no more than 0.03 per cent can be tolerated without some adverse effect. Shell thickness decreased as the dosage of sulfanilamide was increased.

Mattis and co-workers (1946) concluded that therapeutic doses of sulfamerazine at levels of 1 per cent or less in the feed probably would not have any serious toxic effects on the growth of young chickens, but Bankowski (1948) showed that both sulfamerazine and sulfamethazine had a depressing effect on egg production.

Sulfaquinoxaline, on the other hand, according to Cuckler and Ott (1955) and Newberne and Buck (1956) had little, if

any, unfavorable effect on young chickens turkeys or ducks unless fed at levels far above the normally recommended dosage. Sulfiquinoxaline does however act as a stress factor by increasing hemorrhages resulting from a deficiency of vitamin K. Adequate vitamin K as from 5 per cent of alfalfa or other potent source is therefore most important when this drug is to be fed for any extended period.

Sulphur Compounds — Certain organic sulphur compounds such as Arasan used for the chemical treatment of seed grains for the control of plant diseases may become a potential hazard to poultry. If seed grains are fed without previous washing to remove the chemical there is danger that (1) growth will be seriously retarded (Ackerson and Mussehl 1955) (2) egg production will be seriously reduced (Heuser 1956) or (3) the eggs laid will be misshapen and soft-shelled resembling those produced by flocks infected with Newcastle disease or infectious bronchitis (Swanson *et al* 1956).

Other Drugs — Various other drugs which are known to be more or less toxic to fowls but on which information is rather fragmentary include sodium fluoride calcium and magnesium chlorides sodium nitrate thallium acetate carbolic acid salicylic acid ipecac and tartar emetic.

MOLDS AND FUNGI

Musty or moldy grain has long been considered dangerous as food for poultry and numerous cases have been reported in which mortality was supposedly the result of feeding such material. It appears however, that certain samples of grain may be very moldy without being toxic while others which do not appear to be of low quality may cause a high death loss the difference being in the kinds of molds or other organisms present.

Ronk and Carnick (1931) fed young chicks from hatching time to eight weeks of age on rations which contained 20 and 30 per cent of moldy corn and obtained as good growth on these diets as on rations containing corn of No. 2 quality. They concluded that "No apparent deleterious effects resulted when chicks received moldy corn which was infected

with species of *Penicillium*, *Diplodia*, *Fusarium*, *Mucor* or *Rhizopus*, *Aspergillus*, and undetermined organisms including yellow bacteria." In actual practice, however, it would still seem to be wise to avoid the feeding of moldy grain unless preliminary trial with a small group of chicks or hens has shown no harmful effects.

Titus and Godfrey (1934), after a two-year study of the feeding value of barley affected by the fungus *Gibberella saubinetii*, concluded that "scabbed barley whether slightly moderately, or very badly scabbed, may be expected to give essentially the same results as normal barley so far as maintenance of live weight, egg production, and economy of egg production are concerned."

Quigley and Waite (1931) fed hens on wheat that was badly damaged by stinking smut and concluded that, "Wheat damaged by stinking smut is reasonably satisfactory as a poultry food from the standpoint of palatability and the absence of deleterious effects, although its feeding value appears somewhat reduced due to the destructive action of the smut."

"On the basis of this experiment it is not recommended that farmers and poultrymen deliberately purchase smutty wheat for feeding purposes, but it seems evident that such smutty wheat as is produced on the farm or that may be obtained at an attractive price may be fed to poultry as the wheat portion of a scratch mixture, with a reasonable degree of satisfaction."

Horvath (1930) reported that 4 per cent of corn smut, *Ustilago zeæ*, added to the diet for a period of three months was not injurious to hens and did not affect their chemical blood composition or their egg-laying capacity. The eggs were normal in composition as far as could be determined and were not injurious as food.

That fowls may be poisoned by the ergot fungus, *Claviceps purpurea*, has been pointed out by Pearl, Surface and Curtis (1915) and by van Heelsbergen (1929). The fungus is chiefly associated with rye, and since but little rye is fed to poultry in America ergot poisoning is of rare occurrence here. It is said to be more common in European countries where rye is often used in poultry rations.

Forgacs and Carll (1955), in a preliminary report, suggested that toxic fungi may be involved in field cases of hemorrhagic disease

PLANTS AND SEEDS

Certain plants and seeds to which fowls sometimes have access, or which may be included in mixed rations, are poisonous. As already suggested, few of these cause trouble under natural feeding conditions unless the supply of normal green forage is short. The practical means of avoiding trouble from these sources is to see to it that the fowls do not have access to any of the poisonous plants.

Black Locust —The poisoning of mature fowls as a result of eating leaves of the black locust, *Robinia pseudacacia*, was reported by Barnes (1921). He stated that affected chickens lie down apparently paralyzed, pass thin, slimy, greenish feces containing strings of mucus or mucus and blood, and breathe very deeply and heavily with a thumping motion. Locust leaves fed to chickens in the laboratory resulted in the appearance of these symptoms, followed by death in from twelve to twenty-four hours. This report was from West Chester, Pa., and it was suggested that the leaves are toxic only during a part of the year, from about July 1 to the middle of August.

Blue Lupine —According to Massey, Hoffmann and James (1947) as little as 20 per cent of ground seed of the blue lupine, *Lupinus angustifolius*, in a starting ration killed 50 per cent of the chicks to which it was fed. Sub-lethal doses had a progressively retarding effect on growth as the level of the ground seed was increased from 3 to 15 per cent. These authors concluded that blue lupine seed should not, under any circumstances, be included in poultry feeds. Even seed from the recently developed non-alkaloid sweet lupines would be dangerous unless the crop were grown in complete isolation from the high-alkaloid types.

Corn Cockle —The seed of the corn cockle, *Agrostemma githago*, a weed common to wheat fields throughout the world has long been known to be poisonous to animals, and to humans when forming an appreciable adulteration in wheat flour. Quigley and Waite (1931) studied its effects on poultry

and reported their results in some detail. They state that whole cockle seed is very unpalatable to fowls and that it does not constitute a serious problem even when present as a considerable adulteration in a grain mixture for the reason that chickens do not eat whole cockle seed. They found that the palatability of mash mixtures was adversely affected by ground cockle seed even in amounts as low as 0.5 per cent. They reported the toxic dose as about 0.2 per cent of the body weight, and the minimum lethal dose as about 0.25 per cent of the body weight of fowls.

Heuser and Schumacher (1942) observed that chickens affected with corn cockle poisoning presented a listless and unkempt appearance, with rough feathering and diarrhea. Characteristic lesions of cheesy material were present in the mouth and under the tongue. There was depression of both the respiratory rate and the heart rate but body temperature and red blood cell count were not affected. Partly grown chickens developed a tolerance to the poison so that cockle amounting to as much as 0.5 per cent of the body weight could be eaten daily without interfering with growth.

Typical autopsy findings in birds fed lethal doses of pure corn cockle seed are given as the presence of a yellow, caseous lining in the crop, an amber, gelatinous exudate next to the outer muscular coat of the crop, a collection of similar material within the pericardium of the heart, hemorrhages or congested areas in the fatty portion of the heart, a clear fluid around the intestine, and some congestion in the lungs and trachea.

The poisonous material is known as githagin, and has also been referred to as saponin, agrostemmin, sapotoxin and similacin. Heating to 50° C is sufficient to destroy the poisonous principle.

Cottonseed Meal—That cottonseed meal has certain limitations as a protein supplement for poultry has long been recognized but very little work has been done in an attempt to study and measure its toxicity. The poisonous principle in cottonseed is gossypol. Menaul (1923) has shown that gossypol causes death in animals by reducing the oxygen-carrying capacity of the blood. When injected directly into the blood stream its toxic action is immediately apparent.

odor of crushed *crotalaria* leaves which is noted upon opening these organs.

The poisonous alkaloid has been extracted and tested at the Florida Agricultural Experiment Station, and the name monocrotolin has tentatively been proposed for it.

Emmel (1937) studied the pathology of *crotalaria* poisoning and reported that the outstanding lesions are innumerable petechiæ in the serous membranes and visceral fat, as well as a dark liver, marbled in color. He also found *C. retusa* to be about as toxic as *C. spectabilis*, but suggests that a different toxic principle may be involved because of the absence of extensive hemorrhagic lesions in fowls poisoned by the seeds of *C. retusa*.

Since *C. spectabilis* is extensively grown in the southern states as a soil-building legume, it is important that poultry flocks be so managed that there will be no opportunity for the chickens to eat the seed. It is worth noting that several other species of *crotalaria*, viz., *C. striata*, *C. grantiana*, *C. incana* and *C. intermedia*, were non-toxic when force-fed in 5- and 10-gram doses to chickens and quail.

Daubentonia Seed.—That the seeds of the ornamental shrub, *Daubentonia longifolia*, are poisonous to poultry was first reported by Shealy and Thomas (1928). As few as nine seeds caused the death of some birds. The shrub is common in all the Gulf States, and the seed is readily eaten by fowls. Poisoned fowls show a staggering gait, ruffled feathers, drooping wings, purplish comb, muscular twitching and diarrhea. Treatment is ineffective. According to the authors mentioned, the toxic principle is found only in the seed, and poisoning of poultry can be prevented by picking and burning the seed pods before they reach maturity. When the pods are fully mature they split open, liberating the seed. Hence it is important that all pods be destroyed before they mature, unless the poultry flock is so yarded as not to have access to the shrub.

Death Camas.—The death camas (*Zygadenus* sp.) is a well-known cause of stock poisoning and may cause poisoning in poultry under conditions in which no other green forage is available, as in early spring, or after a fall frost which has killed other green plants. Such poisoning is probably rare.

Lille and Bird (1950) fed pure gossypol and pigment glands from cottonseed meal at levels which supplied equivalent quantities of gossypol daily to young chicks and concluded that the toxicity of the pigment glands results entirely from the gossypol which they contain. Hewang and Bird (1955) fed pure gossypol and cottonseed meals containing known amounts of free gossypol to chicks using several different levels and concluded that the free gossypol content of chick diets should not exceed 0.02 per cent if growth depression is to be avoided. Fortunately the cottonseed meal processors are finding ways of making their product safe for poultry feeding either by removing the pigment glands or by inactivating the gossypol.

Coyotillo—The coyotillo plant *Karwinskia humboldtiana* which grows in southwestern Texas and Mexico produces a more or less complete paralysis in chickens as well as in other animals according to Marsh, Clawson and Roe (1928). The effect is peculiar in that symptoms do not ordinarily appear until several days and sometimes as long as three weeks after feeding. This naturally adds to the difficulty of diagnosis. Recovery seldom takes place in severe cases.

The minimum toxic dose for chickens was found to be 0.3 per cent of the live weight fed as dry material (fruit or seed) and it appears that the minimum lethal dose is but slightly larger. No indications of any successful remedy were found in the experiments reported.

Crotalaria Seed.—Thomas (1934) found the seed of *Crotalaria spectabilis* to be toxic for chickens, quail and doves when fed or eaten in considerable numbers. Although quail did not voluntarily eat the seed under either experimental or field conditions, chickens did eat enough of the seed to cause sickness and death. Turkeys were not poisoned by eating as many as 1000 seeds of this species.

Poisoning from this cause may be either acute or chronic depending upon the amount of seed consumed and the resistance of the individual chicken. In the acute type death occurs in from one to ten days but in the chronic type the poisoned birds may linger for months. Diagnosis of the trouble in acute cases can usually be made by the presence of the seed in the crop and gizzard and by the characteristic

odor of crushed *crotalaria* leaves which is noted upon opening these organs

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The growing plant is similar in appearance to the onion, but has no odor. It is common in Nebraska, Kansas, Oklahoma and Texas.

Niemann (1928) reported that a cockerel fed between 5 and 10 grams of ground material from a specimen of the plant developed marked symptoms of poisoning within twelve hours. Diarrhea was an early and persistent symptom, and the droppings were characterized by a peculiar, penetrating, highly offensive odor.

Glottidium Seed — *Glottidium vesicarium* is a common plant in Florida, and fowls running at large may have access to quantities of the seed. Force-feeding of 150 seeds in a single dose proved fatal to adult White Leghorns, according to Emmel (1935). Well-fed birds refused to eat the seed under experimental conditions, while semi-starved birds ate some seed but not enough to prove fatal. A few cases of natural poisoning by these seeds have been observed.

The most consistent macroscopic lesions were necrotic enteritis and necrosis of the bulbous portion of the gizzard.

Milkweed.—Chickens are not likely to eat milkweed except when it is the only forage available to them, and hence the danger of poisoning from this source is rather remote. It is interesting to note, however, that Pammel (1917) reports a case in which about 500 chickens were lost from what appeared to be milkweed poisoning. Campbell (1931) mentions another case in which serious loss occurred among eight-week-old White Leghorn chicks and among mature fowls as a result of eating the narrow-leaved whorled milkweed, *Asclepias mexicana*. Laboratory experiments showed that the roots, as well as the leaves, stems and blossoms, were poisonous.

Stiles (1942) found the whorled milkweed, *Asclepias galioides*, to be very poisonous to turkey poults. Young turkeys fed one gram of milkweed per 100 grams live weight showed spasms within one hour and death within five hours. Immature, tender plants were more toxic than the mature weeds.

Nightshade — A case is reported by Hansen (1925) in which the death of chickens and ducks is attributed to poisoning by black nightshade, *Solanum nigrum*, a common weed of yards

and waste places. The chickens simply became paralyzed and lay stretched out on the ground, unable to make any use of their muscles, and died a short time after. No further trouble was experienced after the nightshade plants were removed from the poultry yard. The berries of black nightshade have been reported as being poisonous to ducks and chickens.

Pokeweed.—Berries of the pokeweed, *Phytolacca decandra*, are often considered to be poisonous, but Hendrickson and Hilbert (1931) were unable to poison chickens by either forced or voluntary feeding of the ripe berries over a period of three weeks. They concluded that the ripe berries are not poisonous to chickens.

Potatoes.—Although potatoes are often used as a stock and poultry food, it appears that under certain conditions they may be dangerously poisonous. Hansen (1927) reports a case in which chickens were fatally poisoned by the feeding of small green sprouts which had been removed from several bushels of potatoes when taken from storage during late winter. The poisonous principle in potatoes is solanin, and it is stated that the solanin content seems to increase to dangerous levels when the tubers turn green under the influence of sunlight.

Tobacco.—The use of tobacco as a vermifuge makes it desirable to have some information regarding its toxicity. Hunter, Haley, and Knandel (1931, 1934) have shown that when tobacco having a high (5 per cent) nicotine content is used, growing chicks can tolerate as much as 0.06 per cent of nicotine in the ration during the third and later weeks without any apparent harmful results. Feeding the same nicotine levels in the form of ground cigar-leaf tobacco having a low (0.86 per cent) nicotine content, retarded the growth of chicks and caused an increase in mortality. Nicotine is one of the strongest of all poisons, but it would seem from these results that other substances in tobacco are also poisonous to chicks.

INSECTS

There are many insect pests which annoy poultry, and many others which serve as intermediate hosts for poultry

parasites, but apparently only one species has been definitely incriminated as being poisonous when eaten by fowls. This one is the rose chafer, *Macrodactylus subspinosus*. Lamson (1916, 1922), Bates (1916) and Gallagher (1920) have reported quite fully on the poisonous character of rose chafers. Lamson was the first to show that the death of chickens following the eating of rose chafers was not the result of mechanical injury. He reported that a water extract of crushed rose chafers was fatal when fed to young chickens of various ages, though mature fowls were not killed by it.

Chickens will feed upon the insects ravenously, and from 15 to 20 rose chafers are sufficient to kill a chicken one week old. From 25 to 45 are necessary to cause the death of chickens three weeks old, while those ten weeks old or older are rarely killed by eating the insects. Death usually occurs within twenty-four hours or the chickens begin to improve.

According to Gallagher, the rose chafer is found in the area from Canada to Virginia, Tennessee and Oklahoma, and from the Atlantic coast to Colorado. There is but one generation produced each year, and the length of life of an individual is about three weeks. The beetles usually appear suddenly in late May or early June and disappear a month or six weeks later. In the northern part of their range these dates are about two weeks later.

OTHER POISONOUS SUBSTANCES

Various other substances, most of which do not fall logically into the foregoing classification, have been known to cause poisoning of fowls. Tully and Franke (1934) have reported a type of poisoning resulting from the use of corn, barley and wheat grown in certain limited areas in central South Dakota.

Subsequent work at the South Dakota Agricultural Experiment Station has shown that the toxic effects are the result of selenium in the "alkali" grain. When the toxic grains were fed at levels which furnished 15 parts per million of selenium in the ration, laying hens lost weight, egg size was reduced, and hatchability decreased to zero.

No normal chicks hatched from eggs laid after the seventh day of selenium feeding. Conversely, when the hens were changed back to a diet of normal grain the toxic effects of selenium disappeared within a week. With 5 parts per million of selenium in the ration, hatchability was not appreciably affected, though there was some evidence of selenium poisoning.

Bayon (1934) has called attention to the dangers which may arise from the addition of antiseptics to the drinking water, especially in the case of young chicks. He expresses the opinion that water is such an essential part of every living creature's existence that the addition of poisons to it can rarely be justified. He further says that he has far too frequently seen stunted, undersized pullets whose bowels were scoured, thin and lifeless as the result of corrosive sublimate, permanganate of potash, iodine or hydrochloric acid having been added to the drinking water.

Fumigants are occasionally met with in the form of poisons. The increased use of formalin in incubator fumigation has led to the occasional accidental poisoning of chicks or hens. While this drug in the strength recommended for incubator fumigation is apparently not harmful to newly-hatched chicks, it is exceedingly poisonous to older chickens.

Hydrogen sulphide would not often be encountered as a poison, but Clough (1930) reports that it is poisonous either in the air breathed or in solution in the drinking water. He states that 1 part in 1500 of air is fatal to birds.

Poultry workers are confronted annually with a great many cases in which some proprietary feed mixture, or some ingredient in a home-mixed ration is suspected of having caused the death of chicks or older fowls. It is perhaps only natural to blame the feed when no other cause of death is readily apparent, but examination of suspected feeds in a number of laboratories over a period of several years has revealed a negligible number of samples that were actually poisonous. Quigley and Waite (1931) reported that of 61 samples examined over a period of three years, and tested by actual feeding trials, not one was found to kill healthy birds.

Halpin (1934) states that in the routine testing, over a

period of several years, of numerous samples of feed suspected of poisoning chickens, only two have been found which proved actually to be poisonous. A third sample was suspicious. The exact number of samples tested is not available, but the total in one of the years covered by the report amounted to 165.

In spite of the rather extensive list of poisons to which reference is made in this chapter, the fact remains that actual poisoning of poultry is of relatively rare occurrence, and when poisoning does occur it is still more unlikely that the source is to be found in either ready-mixed feeds or in the ingredients commonly sold for use in home-mixed rations.

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Chapter 12

INTERNAL PARASITES

DOMESTIC fowls are susceptible to infestation by a large number of internal parasites, and it is safe to state that very few flocks are entirely free from them. The damage done by these unwelcome pests, both directly and indirectly, cannot be accurately calculated, but it is undoubtedly of such magnitude as to constitute one of the major problems confronting the poultry industry.

Worms are prolific and ever ready to establish themselves in susceptible hosts, and failure to institute appropriate measures of control will lead, sooner or later, to decreased production, lowered returns, and in many cases to a high death loss. The devitalizing effect of parasites is an important factor in rendering fowls more susceptible to disease.

The symptoms caused by worm infestation vary and are not constant enough to be relied upon for diagnosis. In heavily parasitized young birds the common manifestations are stunted growth, emaciation, weakness and death; in laying hens, production is lowered or entirely stopped. The symptoms are more severe and the death-rate higher in young birds than in older fowls.

The changes observed at autopsy depend upon the kinds and numbers of parasites present. From the standpoint of diagnosis, the lesions encountered are of far less value than the finding and identification of the causative parasites themselves.

New organic materials which are specific for the control of certain internal parasites have recently been developed and more may be expected, but it is still true that the most effective method of dealing with the problem is to apply the principles of good sanitation and clean management. Well-managed flocks do not often suffer from severe infestations of the more common parasites. Adequate separation of young and old stock, systematic rotation of range areas, and proper disposal of manure and trash which may provide shelter and

breeding places for intermediate hosts will greatly reduce the chances of infestation. Control of house flies and stable flies is also important.

There are two principal types of internal parasites of poultry, viz., roundworms and tapeworms. A third group is made up of flukes and, although they are not so frequently encountered at present, their incidence appears to be on the increase.

LARGE INTESTINAL ROUNDWORMS

The large roundworm *Ascaridia galli* known to nearly every flockowner is a common parasite of chickens, and less often of turkeys, ducks and geese. A similar worm, *Ascaridia columbae*, is frequently found in considerable numbers in pigeons. The worm as seen in chickens is from $1\frac{1}{2}$ to 4 inches in length and is usually found in the small intestine. Not infrequently the parasites are so numerous as completely to plug that portion of the bowel inhabited by them. It is this worm which is occasionally found in an egg having wandered up the oviduct from its normal intestinal location with subsequent incorporation in the descending egg. Fortunately, however, such an occurrence is rare.

Life History—The reproductive cycle of the large roundworm is direct. The eggs, microscopic in size, are deposited by the female worm in the intestinal contents and are subsequently passed to the outside in the droppings of the bird. When first passed out the eggs are not infectious, but under favorable conditions of warmth and moisture they embryonate and become infectious in from ten to sixteen days. During this period of embryonation a small coiled worm has developed within the egg and if the latter is eaten by a susceptible fowl the young worm is liberated from the shell and begins development in the intestinal tract of the host. In a few days the young worms penetrate the lining of the bowel and undergo further development, after which they emerge into the intestinal canal and continue their growth. A period of fifty to sixty days is required for the young worms to attain maturity, at which time the females are capable of laying eggs and starting the cycle over again.

The eggs, in common with most parasite ova, possess considerable resistance to climatic influences. Ackert and Canthen (1931) found that eggs exposed in less than $\frac{1}{2}$ inch of unshaded soil were killed in three weeks, but when shaded at the same depth they survived from spring to autumn.



FIG. 70.—Section of intestine showing roundworms protruding from a cut end (Courtesy of California Agricultural Experiment Station)

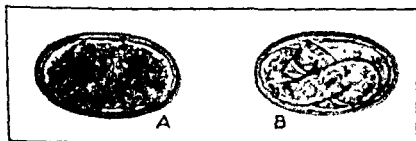


FIG. 71.—1 eggs of the roundworm, *Ascaris suum* as they appear under the microscope. A, An egg in the non-infectious stage when passed from the intestinal tract of the chicken, B, an infectious or embryonated egg, showing the young coiled worm inside.

Susceptibility—The damage done to fowls by infestation with *Ascaridia galli* is greatest in young birds. It appears that several factors may influence the degree of infestation and the severity of the ensuing results. Ackert and associates (1935) found that the heavy strains and varieties of chickens are more resistant to infestation than are the lighter ones. Using the average worm length per group of chickens as the criterion, Ackert, Porter and Beach (1935) noted in several groups of chickens that age is an important factor in resistance to growth of the nematode. Starting with forty-five days of age significant differences were observed up to ninety-three days of age at which time the maximum resistance appears to be reached. These authors offer as the most plausible explanation the probability that as the chicken grows older there are developed in its body growth-inhibiting factors which react against the development of the worms.

Frick and Ackert (1948) have shown that the mucus of the intestinal tract of fowls contains a heat-stable factor which is inhibitory to *Ascaridia galli* and that the efficacy of such mucus as a worm-growth inhibitor varies directly with the age of the fowl. Larger amounts of mucus are present in older than in younger fowls and this may well account, in part at least, for the increased resistance of older birds to infestation by the ascarids.

Symptoms—General unthriftiness, as manifested by variable appetite, emaciation, retarded growth and weakness is commonly observed in young parasitized birds, while loss of condition, weakness and lowered production are characteristic of heavy infestation in laying fowls. Ackert and Herrick (1931) attribute the severe effects of infestation by the parasite in young chicks to injury of the intestinal wall, loss of blood, probable bacterial infection, absorption of metabolic waste from the worms and loss of weight from impaired appetite. Ackert (1930) also lists loss of lymph, retarded bone and muscular development, reduction in the amount of blood sugar and diminution in the size of the thymus gland as additional detrimental effects induced by the presence of these parasites.

Prevention.—General measures of sanitation, designed to prevent development of the eggs and their subsequent inges-

tion are the most effective means of controlling the spread of roundworm infestation. Low wet areas in the yards and runs should be filled, and adequate drainage provided. It is particularly important that the damp spots which are commonly found around the watering devices be eliminated since moisture greatly facilitates development of the worm eggs.

Cultivation and rotation of lots and runs are of value in reducing the number of infectious eggs to which the fowls will have access. Removing and burning all litter at intervals of not to exceed ten days will prevent any worm eggs contained in it from developing to the infectious stage. The water and feed troughs should be thoroughly cleaned and disinfected and then so placed as to prevent subsequent contamination by droppings.

Young chicks, because of their great susceptibility to infestation, should be reared apart from older birds until they are at least four months old, after which time they are somewhat more resistant.

Treatment.—For removal of the large roundworm, Buckley, Bunyea and Cram (1935) recommend carbon tetrachloride or tetrachlorethylene. Both are powerful agents and care must be exercised to prevent overdosing. The treatment is administered by giving a dose of 1 cc. to each mature fowl, either in a gelatin capsule or by means of a small flexible rubber tube introduced directly into the crop. In treating young fowls the dose must be reduced in proportion to the size of the bird.

Ackert and Graham (1935) studied the efficacy of carbon tetrachloride for removing *A. galli* from chickens. When used at the dose rate of 4 cc. per kilogram of body weight on chickens ten weeks of age all nematodes were removed and practically no toxic effects were observed. When the drug was given at the dose rate of 10 cc. per kilogram of body weight all worms were removed but there was a mortality-rate of 25 per cent in the chickens. The egg production of pullets was materially reduced for a period of seven to ten days following administration of the drug in doses of 4 cc. per kilogram of body weight. When used in doses of 6, 4, and 2 cc. per kilogram of body weight, the drug tetrachlorethylene was found to be efficacious in removing the worms

but exerted a more toxic effect upon the chickens than did carbon tetrachloride

Harwood and Guthrie (1944) demonstrated a good degree of effectiveness of a mixture containing phenothiazine and nicotine-bentonite for the removal of ascarids from chickens. The mixture consisted essentially of 1 part of phenothiazine and 2 parts of nicotine-bentonite (with a nicotine content of 5 per cent) and tablets weighing 1.33 grams were administered to the birds.

Jaquette and Wehr (1949) used a nicotine-bentonite and phenothiazine mixture added to a regular dry mash and found that it removed a very high percentage of both ascarids and cecal worms within four days. The medicated mash contained approximately 0.075 per cent of nicotine sulphate, 0.75 per cent of phenothiazine and 1.4 per cent of bentonite.

Various piperazine compounds, especially piperazine adipate and piperazine citrate, have recently been shown to be highly effective against *Ascaridia galli* either in single doses of 300 to 500 milligrams per kilogram of body weight, or when added to the drinking water at the rate of 2 to 4 grams per gallon. The anthelmintic action of these compounds depends on their ability to produce a state of narcosis in the worms which are then carried out in the normal flow of intestinal contents (Horton-Smith and Long, 1956). There is some question as to whether these drugs have any appreciable effect on tissue phase larvæ of the parasite.

During the period following administration of vermifuges the treated birds should be confined so that the worms and eggs which are expelled may be gathered with the litter and destroyed by burning.

CECAL WORMS

The cecal worm, *Heterakis gallinae*, is found as a common inhabitant of the ceca of chickens, turkeys, guineas, ducks, geese, peafowls, partridges, grouse and quail. Schwartz (1924) reported the presence of a closely allied cecal worm, *Heterakis wolonche*, in pheasants. The cecal worm of chickens is small, being but $\frac{1}{4}$ to $\frac{1}{2}$ inch in length, and is grayish-white in color. The parasite is frequently found in great

numbers and may produce severe inflammation of the ceca, particularly in young birds. The important part which the eggs of these worms play in the transmission of blackhead has been discussed under that disease in Chapter 8, page 237.

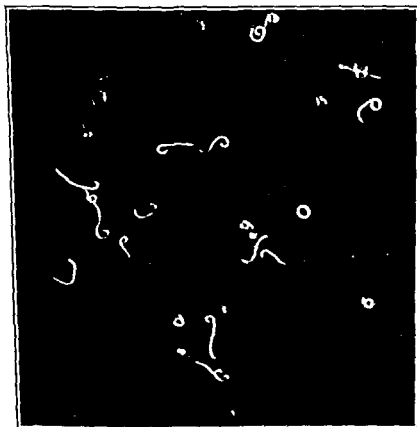


FIG. 72.—Cecal worms of poultry, *Heterakis gallinae*. (Courtesy of Illinois Agricultural Experiment Station.)

Life History.—The life cycle of the cecal worm is direct. The eggs, deposited in the ceca by the female worms are voided with the droppings of the bird. Under conditions of sufficient warmth and moisture the eggs embryonate and become infectious in from seven to fourteen days. The eggs are not readily destroyed by climatic influences, as was shown by Graybill (1921), who found that fully developed eggs, kept in soil out of doors under natural conditions, contained living embryos after a period of eight months.

Upon being swallowed by a susceptible fowl, the young worms are freed from their shells and penetrate the lining of the ceca. During their development in the cecal walls the young parasites may inflict severe damage to the tissue, after which they return to the lumen of the ceca to complete their growth. About sixty five days are required for the entire cycle from egg to mature worm to be completed.

At least two species of dung earthworms common in barn lots, especially in horse manure have been found by Scott (1913) and Ackert (1917) to be capable of transmitting the cecal worm, although such a mode of transmission is not necessary to the life cycle of the parasite. The eggs may be eaten by the earthworm and later passed out, to be picked up by fowls, or the earthworms may be eaten by fowls and the eggs thus transmitted.

Susceptibility—It was found by Clapham (1933) that when chickens four months of age were fed a ration deficient in vitamin A they were no more susceptible to infestation by the cecal worm than were birds of the same age given a ration containing an adequate amount of the vitamin. The same author (1934), however, found that young chickens deprived of an adequate amount of calcium and phosphorus were more heavily infested by *H. gallinæ* than were those which had received a sufficient amount of these minerals. In further studies upon the susceptibility of chickens to the parasite Clapham (1934) concluded that infestation at an early age does not appear to induce any resistance to later infestations. This worker also found that chickens of all ages appear to be fairly susceptible although some resistance was noted in older fowls.

Symptoms—The irritation caused by these small worms in the ceca often leads to general unthriftiness, and in young chicks death may result from heavy infestation.

Prevention.—Since the life cycle of the cecal worm is practically identical with that of the large intestinal roundworm, the measures suggested for control of the latter are applicable to the cecal worm. General sanitation, rotation of lots and yards, and strict separation of young from old birds constitute the basis for preventing spread of the parasite.

Treatment.—McCulloch and Nicholson (1940) have reported favorably upon the effectiveness of phenothiazine against *Heterakis gallinæ*. They administered the drug to chickens carrying a heavy natural infestation of cecal worms, using doses ranging from .05 gram to .5 gram. The drug was given individually in capsules as well as in a flock treatment. Repeated doses to individual birds were found to be only slightly more effective than flock medication. The average effectiveness was between 95 and 100 per cent, both as to killing the worms and their expulsion. Enormous doses appeared to have no harmful effect upon the chickens, indicating a wide range of safety, and no appreciably undesirable effect upon egg production was noted following individual dosing. There were no digestive disturbances except a slight softening of the feces twenty-four after administration and even massive doses did not impart any undesirable flavor to the meat.

Harwood and Guthrie (1944) and Jaquette and Wehr (1949) found the phenothiazine and nicotine-bentonite mixture as previously described under treatment for *Ascaridia galli* also effective against cecal worms.

CAPILLARIA WORMS

These important parasites, sometimes called hairworms, are found in the crop, esophagus, small intestines and ceca of chickens, turkeys, crows, grouse, quail, ducks, pigeons, partridges and pheasants. Three of the most important species are *Capillaria contorta*, *Capillaria columbæ* and *Capillaria annulata*. They are from $\frac{1}{2}$ to $\frac{3}{4}$ inch in length, colorless, exceedingly fine and hair-like, and because of their small size are difficult to detect with the unaided eye. The worms sew themselves into the lining membrane of the invaded organ and in heavy infestations cause marked inflammation and thickening of the wall.

Life History.—*Capillaria contorta* and *Capillaria columbæ* have direct cycles of reproduction similar to that of the large roundworm and the cecal worm, in which the eggs of the parasite are passed in the droppings to the outside where they develop to infectiousness. The eggs require a considerable amount of moisture for development and Cram (1926) stated

that drying for a period of three to four weeks destroyed them. In the case of *Capillaria annulata* it has been found that at least two species of earthworms may serve as intermediate hosts, the cycle thus being indirect.



FIG. 73 — A capillaria worm enlarged about 15 times. Note the eggs in the central section. (Courtesy of Illinois Agricultural Experiment Station.)

Symptoms — Birds heavily infested with capillaria worms show unthriftiness, weakness, diarrhea, listlessness and loss of weight. They sit huddled on the ground, the feathers are ruffled, the vent is soiled, and the visible mucous membranes are pale. In some cases weakness of the neck muscles causes the affected bird to lose control of the head, but this is not a constant symptom.

Prevention — The same hygienic measures suggested for the control of similar parasites are applicable to capillaria worms.

Treatment — Carbon tetrachloride administered in 1 cc doses has been found to be effective against some species of

hairworms. To obtain the best results the treatment should be repeated in seven to ten days.

Emmel (1939) obtained favorable results in turkeys infested with *C. contorta* by feeding 5 per cent of commercial flowers of sulphur in the regular mash. Improvement was noted in many affected birds within a few days and continued during the trial period of three weeks. Recovery occurred in all birds which were able to eat when the treatment was started.

OTHER INTESTINAL ROUNDWORMS

Of much less frequent occurrence is the extremely small parasitic roundworm, *Strongyloides avium*, which inhabits the ceca and small intestines of chickens. The parasites cause thickening of the cecal walls and, in severe cases, bloody diarrhea may be present. Young chicks may be seriously affected, but if they survive the effects of early infestation they usually show no symptoms when grown, even though they continue to harbor the parasites. No treatment has been devised for this parasite.

A serious disease of pigeons is caused by infestation with the roundworm known as *Ornithostrongylus quadriradiatus*. Turtle doves and mourning doves are also reported to be susceptible to infestation. It is a very slender, hair-like parasite which inhabits the small intestine. The worm is a blood-sucker, and pigeons infested by it show diarrhea and great emaciation. The death-rate in some cases is very high. Cram and Cuvillier (1934) were unable to obtain consistent results with tetrachlorethylene or with thymol when used as anthelmintics, but state that maintenance of conditions which result in drying of the droppings of infested pigeons appears effective in preventing spread of the parasite. They were not able to induce experimental infestation in the chicken, turkey, guinea fowl or domestic duck.

GIZZARD WORMS

Four different species of gizzard worms have thus far been reported in birds in this country; viz., (a) *Cheilospirura hamulosa* in chickens and turkeys, (b) *Cheilospirura spinosa*

in ruffed grouse and quail (c) *Amidostomum anseris* in domesticated ducks and geese, and (d) *Acuaria anthuris* in crows

The gizzard worm of chickens is from $\frac{1}{2}$ to $\frac{3}{4}$ inch in length, fairly slender, and of a reddish color. These parasites burrow through the tough, horny lining of the gizzard and bury themselves in the heavy muscles of the organ near the entrance of the proventriculus, causing the formation of nodules on the surface of the gizzard. The eggs are passed out into the gizzard through the holes made by the burrowing process in the horny lining and pass along with the contents to the outside.

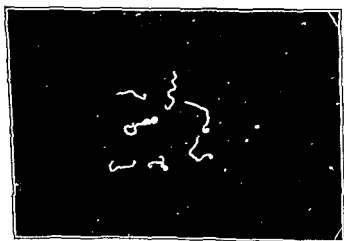


FIG. 4.—Gizzard worms *Cheilosporurum hamulosa*. Natural size. (Courtesy of Illinois Agricultural Experiment Station.)

This worm requires an intermediate host for completion of its life cycle, and grasshoppers, weevils, sandhoppers and several species of beetles have been found to serve in this capacity. The worm eggs after being passed in the droppings of the bird are ingested by the intermediate host. The small worms (larvæ) which hatch from the eggs undergo development in this host and become infectious in from twenty-two to sixty-seven days. If the intermediate host is eaten by a susceptible bird the larvæ are set free and grow in

the body of the fowl, reaching maturity in about seventy-six days.

The gizzard worm does not produce any definite symptoms in fowls, but heavy infestations interfere with the functioning of the gizzard, causing general unthriftiness, emaciation, anemia, and in some cases death.

To prevent ingestion of the worm eggs by grasshoppers it is essential that the litter and droppings be removed frequently and disposed of by burning. To reduce the number of grasshoppers eaten by the birds it has been recommended that the fowls be confined to runs which have been sown to short, thick vegetation rather than to allow them to run in tall, dry vegetation which is favorable to grasshoppers.

So far as is known, no treatment has been proved to be efficacious against gizzard worms. Carbon tetrachloride and tetrachlorethylene in appropriate dosage (1 cc. to a mature fowl) have been suggested as having potential remedial value, but the location of the parasites in the wall of the gizzard would appear to render attempted medication futile.

The gizzard worm of ruffed grouse and quail, *Cheilospirura spinosa*, has been reported from several states in the eastern part of the country. The worm is very similar in appearance to the gizzard worm of the chicken, and the life cycles of the two parasites are practically the same, both requiring grasshoppers as intermediate hosts. Heavy infestation results in extensive damage to the wall and lining of the gizzard.

The gizzard worm of domestic ducks and geese, *Amidostomum anseris*, is a small, slender roundworm which burrows in the horny lining of the organ. The worms are capable of inflicting severe damage to the gizzard and their presence is often indicated by the presence of rough, brownish areas on the lining of the organ.

The life cycle of this parasite is direct. The eggs, after being voided in the droppings of the bird, hatch into young worms which are in turn picked up by fowls. The young parasites then find their way to the gizzard, in which they develop to maturity.

Dullness, loss of appetite, emaciation, as well as difficulty in eating and drinking may be manifested by affected birds.

Buckley, Bunyea and Cram (1935) state that 1.5 to 2 cc.

of carbon tetrachloride given in about 8 cc of liquid cereal has proved of value in removing this parasite from young geese

SPIRAL STOMACH WORMS

Among the less common parasitic roundworms is *Dispharynx spiralis*, a short, thick, coiled worm which inhabits the proventriculus of chickens turkeys pigeons, guinea fowls, and certain wild birds. These worms are not of frequent occurrence, but when present are capable of producing severe damage to the glandular stomach, where they attach themselves by burying their heads deeply in the wall. Tumor like swellings are usually found at the site of attachment of these parasites and in heavy infestations the wall of the organ becomes greatly thickened and often ulcerated.

Life History—Two species of sow bugs (pill bugs) have been found to function as intermediate hosts for the spiral stomach worm. The eggs of the parasite, after being voided in the feces of the bird, are eaten by the sow bug, in the body of which they develop to infectiousness in about twenty-six days. If the bug is then eaten by a susceptible fowl, the larvæ are liberated and develop to maturity in about twenty-seven days.

Symptoms—Fowls heavily infested with the spiral stomach worm show droopiness and loss of weight despite a ravenous appetite.

Prevention.—Frequent disposal of droppings and litter is necessary to prevent ingestion of the worm eggs by sow bugs. All trash, debris and other material which might furnish hiding places for the intermediate hosts should be removed from the yards and runs.

Treatment.—No specific treatment is known, but it has been suggested by workers in the U. S. Department of Agriculture that carbon tetrachloride or tetrachlorethylene may be of value.

TETRAMERE STOMACH WORMS

The chicken stomach worm, *Tetrameres americana*, is a parasite which occurs primarily as an inhabitant of the

proventriculus of chickens, but it has also been found in bob-white quail. Durant and Knight (1941) have recently reported finding the parasite in eastern cardinals in Missouri. A striking departure from the shape and color usually noted in roundworms is seen in the case of the female tetramere worm. Both sexes of the worm enter the glands in the lining of the proventriculus and grow to maturity in that location. During development, however, the accumulation of eggs causes the body of the female to swell, so that it appears globular in shape; its color also becomes a bright red. The males retain their slender form throughout life.

Life History.—Grasshoppers and cockroaches act as intermediate hosts for this worm. The eggs develop to infectiousness in about forty-two days after being eaten by the intermediate hosts. After the grasshopper or cockroach is eaten by a susceptible fowl, about twenty-five days are required for the worms to develop to maturity.

Symptoms.—The presence of this parasite is not always indicated by symptoms, but in young chickens emaciation and sometimes death result from heavy infestation.

Prevention.—To avoid infestation with this parasite, young chicks should be prevented from eating grasshoppers or cockroaches which may have ingested the droppings of older infested fowls. This is most easily accomplished by complete separation of young and old stock.

Treatment.—No effective treatment has been found, but beneficial results have followed the use of soft, highly nutritious feeds in place of hard grain.

EYEWORMS

The eyeworm of poultry, *Oxyuris mansoni*, is a slender, round, colorless parasite, about $\frac{1}{2}$ inch in length, found under the nictitating membrane or "third eyelid" of chickens, turkeys and peafowls. It may also be transmitted by artificial means to several species of wild birds. The parasite has been reported from Florida and Louisiana in this country.

Life History.—An intermediate host is necessary for the eyeworm to complete its life cycle and the cockroach, *Pyrenoscclus surinamensis*, has been found to serve in that

capacity This cockroach is found under old boards and trash which may be present on the premises

The eggs of the worm in the eye are passed down the tear duct of the bird to the mouth, are swallowed, and pass to the outside in the droppings The eggs or newly hatched larvæ are picked up and eaten by the cockroach, in the body of which they develop for some time When the cockroach is eaten by a susceptible fowl the young worms are released, probably in the crop, and quickly make their way back up the esophagus to the mouth and thence by way of the lacrimal duct to the eye The worms have been found in the eyes of chickens within twelve to twenty-five minutes after the birds had been fed cockroaches containing the young parasites About three months are required for the completion of the life cycle

Symptoms—The irritation produced by the worms in the eye causes the affected fowl to scratch the eye or rub it over the body feathers The eyes are swollen and inflamed and the bird winks continually In some cases the inflammation may be so severe as to lead to destruction of the eyeball

Prevention.—Any trash or debris which might harbor the cockroach should be removed from the yard General sanitary measures, particularly the frequent removal of droppings to prevent their ingestion by cockroaches, are important aids in prevention

Treatment.—The treatment developed at the Florida Agricultural Experiment Station is as follows Instill into the eye a few drops of butyn (local anesthetic), lift the nictitating membrane and place 1 or 2 drops of a 5 per cent solution of creolin directly on the worms Immediately after instilling the creolin the eye must be well irrigated with clean water The worms are instantly killed by the creolin and may then be removed

GAPEWORMS

The fowl gapeworm, *Syngamus trachea*, is a small, reddish roundworm frequently found in the trachea of chickens, guinea fowls and turkeys, also in many wild birds including blackbirds, starlings, magpies swifts, robins, sparrows, lin-

nets, pheasants, partridges and rooks. The male worm is small, being but $\frac{1}{16}$ to $\frac{1}{4}$ inch in length, while the female is much longer, measuring from $\frac{1}{4}$ to $\frac{3}{4}$ inch. The male worm is permanently attached to the female, giving the pair a Y-shaped appearance. This parasite produces the condition



FIG. 75.—Trachea pinned open to show gape worms. (Courtesy of Maryland Agricultural Experiment Station.)

commonly known as "gapes" because infested chickens "gape" and show difficulty in breathing.

Life History—Because of the permanent closure of the vulva of the female worm by the attached male, the eggs can be liberated only by rupture or disintegration of the female. This may occur in the trachea of the bird or after the worms are expelled to the outside. Depending upon favorable conditions of warmth and moisture embryos develop in the eggs in from one to seven weeks and in some cases hatch out. Either the eggs containing the embryos, or the young worms already hatched may be eaten by fowls. After being taken into the digestive tract of the bird, the young worms migrate to the lungs and thence to the trachea, where they mature within seven to ten days.

Earthworms have also been found to play an important part in the dissemination of gapeworm infestation. The eggs or newly hatched larvæ of the parasite are eaten by the earthworms, in the bodies of which they may remain as infectious larvæ for some time. When the worms containing the larvæ are eaten by a susceptible fowl the young larvæ are freed and begin their migration to the respiratory tract. This manner of dissemination however, does not make the earthworm necessary for transmission since as has already been pointed out, direct infestation may also occur. The protection afforded the young worms in the body of the earthworm serves to keep the larvæ alive much longer than when they are exposed to natural climatic conditions, and the young parasites may thus be carried safely through conditions which they would not otherwise survive.

Clapham (1934), by using earthworms as intermediate hosts, has reported success in transmitting gapeworm infestation to chickens from pheasants, partridges and rooks. Certain snails and slugs have been found by Taylor (1935) to also be capable of acting as intermediate hosts for the parasite, and Clapham (1939) states that three arthropods viz., a certain centipede, the "leatherjacket," and the "springtail" have been incriminated as vectors.

Symptoms—It is in young birds under eight weeks of age that gapeworms produce the most serious results. Heavily infested older fowls show only mild symptoms or none at all.

In young subjects, the activities of the worms cause inflammation of the trachea with the accumulation of considerable amounts of mucus. The resultant interference with respiration causes the affected birds to stretch the neck and "gape" with open mouth for air. The birds sneeze, cough, and frequently shake their heads in an effort to expel the worms and mucus from the windpipe. There is impairment of appetite, progressive emaciation and between paroxysms of coughing the birds sit huddled quietly on the floor with the head drawn back against the body, and the eyes closed.

Wehr (1937) called attention to the fact that nodules, caused by the attachment of the male worms, are frequently found on the walls of the tracheas of poults, while such nodules are seldom seen in chicks. These swellings may become so numerous and of such size as to add to the difficulty in breathing.

Wehr (1939) has shown that, contrary to previous opinions, it is in young turkeys rather than chicks that the severest results occur. In poults the symptoms appear sooner, the number of worms is greater, deaths start earlier, and the mortality is heavier than in chicks. He also states that the turkey, rather than the adult chicken, which seldom harbors gapeworms, is an important factor in disseminating gapeworm infestation, and that turkeys and contaminated soil are two of the chief factors in perpetuating the worms from season to season. It has also been shown by other workers that snails and slugs can retain viable larvæ of gapeworms in their bodies for long periods of time, thus furnishing a ready source of infestation to susceptible fowls and serving to perpetuate the worms from one year to the next.

Prevention.—Frequent removal of litter to prevent accumulation of worm eggs and larvæ is important. Because of the part which earthworms and other intermediate hosts may play in transmission of the parasite, all conditions of moisture and shelter which favor their presence should be corrected. Wehr (1939) states that the starling, recently imported into this country may, if it becomes established in areas where gapeworms are prevalent, serve to increase the prevalence of the parasites.

Treatment —Many remedies have been tried against gape worms but until recently they have in general proved unsatisfactory. Clapham (1935) has reported favorable results from the administration of either garlic oil or the synthetic product, allyl sulphide, with the latter preferred because of being cheaper and less pungent. The drug was given by forced feeding with a pipette, the dose ranging from $\frac{1}{2}$ minims to 3 minims of either a 50 per cent or a $33\frac{1}{3}$ per cent solution in linseed oil. The effects are described by the author as marked, and heavily infested birds ceased to gape during the first day of treatment. Within three days the parasites were loosened and coughed up, and in addition, the action of the drug was sufficiently powerful to render eggs of the parasite sterile.

Wehr, Harwood, and Schaffer (1939) tested the efficacy of many drugs and chemicals against gape worms, some of the materials being fed, some injected intratracheally, and others used as fumigants. Insufflation was used in testing many substances, most of them having no beneficial effects whatever, while others showed such low degrees of effectiveness as to render them definitely impractical.

However two of the drugs viz., calcium antimonyl tartrate and barium antimonyl tartrate gave indications of effectiveness and since the latter was less toxic, it was tested further. Infested chicks were placed in covered jars so equipped that the powder on the floor of the jar could be circulated by means of a tube attachment. The powder was dispersed two to four times during a ten minute treatment of the birds, which were then removed, observed and later autopsied. The average efficacy of the drug in 10 experiments involving 143 birds was over 98 per cent. The treated chicks manifested increased coughing and were slightly depressed for a few hours, after which definite improvement was observed, with the birds rapidly returning to normal behavior.

TAPEWORMS

Tapeworms are flat, white, segmented, ribbon like parasites, several species of which are capable of infesting fowls. They vary in size from those barely visible to the unaided

eye to forms having a length of 8 to 10 inches, and are usually found in the first half of the intestinal canal. So far as is known, all tapeworms of the fowl require intermediate hosts for completion of their life cycles. Many of the reproductive cycles have been definitely established, but some still remain to be worked out.

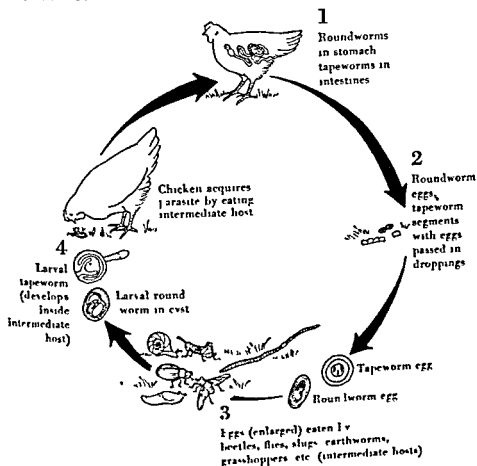


FIG 76—Showing the various stages in an indirect life history of a tapeworm and a roundworm of poultry (From the U. S. Department of Agriculture)

Life History.—The heads of tapeworms are equipped with hooks and suckers by which they attach themselves to the lining of the intestines. The head is the smallest but oldest portion of the parasite. New segments continue to form just behind the head and only the terminal segments or sections are mature and contain fertilized eggs. As the tapeworm

grows, the end segments are detached, pass out in the droppings of the fowl and may be eaten by a variety of beetles, flies, snails, worms and other forms capable of acting as intermediate hosts, in which the eggs develop to the infectious stage. Upon being taken into the body of the fowl when the intermediate host is ingested, the young tapeworms are freed and develop to maturity.

Because of the similarity in general structure and life histories of the fowl tapeworms, a separate description of each is not given here. In the following list (p. 334) of fowl tapeworms, and species of birds infested, the asterisk is used to designate those cases in which further evidence is required to establish the correctness of the intermediate host listed.

Symptoms—As is the case with most parasites, tapeworms produce the severest damage in young fowls. Birds infested with tapeworms present general symptoms of unthriftiness. Droopiness, ruffled feathers, diarrhea, weakness and paleness of comb and wattles are commonly observed. Many affected fowls become weak or even completely paralyzed in one or both legs. In some cases the neck muscles appear to be affected and the fowl twists the head and neck around into unnatural positions.

The intestinal wall of heavily parasitized birds is often thickened and shows catarrhal inflammation. One of the tapeworms, *Raillietina echinobothrida*, produces small nodules in the wall of the intestine and it is these lesions which are occasionally mistaken for tuberculous nodules.

The exact manner in which tapeworms produce harmful effects is not known, but the inflammation of the intestine no doubt interferes with digestion in varying degrees. It has also been fairly well established that the worms produce substances during their growth which are harmful when absorbed by the fowl. In heavy infestations the bowel may be practically occluded, and the normal movement of intestinal contents greatly disturbed.

Prevention.—The droppings which may contain "ripe" segments of tapeworms should be disposed of in such a manner as to prevent the various beetles, bugs, flies, earthworms, slugs and snails from ingesting them. All boards, trash and other unnecessary objects which might serve as

hiding places for the intermediate hosts should be removed from the houses and yards. Elimination of wet areas, and discing of the yards at frequent intervals are helpful in reducing the number of intermediate hosts to which the fowls might have access.

Tapeworms.	Hosts	Intermediate hosts
<i>Raillietina cesticillus</i>	{ Chicken Turkey Guinea fowl Quail	Beetles House flies
<i>Choanotaenia infundibulum</i>	{ Chicken Turkey Guinea fowl Duck	Beetles House flies
<i>Raillietina tetragona</i>	{ Chicken Turkey Quail Peafowl Guinea fowl	Ants
<i>Raillietina echinobothrida</i>	{ Chicken Turkey Pigeon	Ants
<i>Hymenolepis carioca</i>	{ Chicken Turkey Quail	Stable flies Beetles
<i>Hymenolepis cantaniana</i>	{ Chicken Peafowl Turkey Quail	Beetles
<i>Raillietina magninumida</i>	{ Pheasant Guinea fowl	Beetles
<i>Davainea proglottina</i>	{ Chicken	Slugs and land snails
<i>Davainea meleagridis</i> *	{ Turkey	Unknown

Treatment.—Various drugs such as areca nut, thymol, pomegranate root bark, fluid extract of male fern, turpentine, carbon tetrachloride, tetrachlorethylene, oil of chenopodium, rotenone, arecoline hydrobromide, santonin, betanaphthol, hexylresorcinol and pumpkin seed have all been tried for the removal of poultry tapeworms, but none has proved satisfactory.

Harwood and Guthrie (1940) experimentally tested 223 substances and 27 mixtures for efficacy in removing tapeworms from chickens. This list included practically all drugs which have ever been recommended as treatments and in

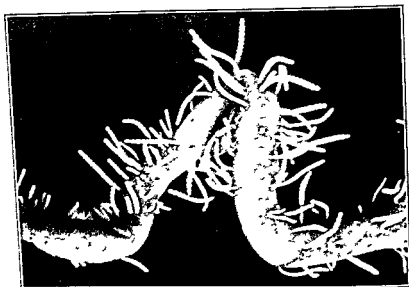


FIG 77 —Inside of a hen's intestine showing attached tapeworms
(*Courtesy of California Agricultural Experiment Station*)



FIG 78 —Section of intestine stretched under water as an aid to finding small tapeworms. About twice natural size.

addition many less common substances. Of this large number, only one, lead arsenate, showed any promise as a poultry tæniacide and it was found to be too toxic to the birds to be recommended for general use.

Guthrie and co-workers later reported that certain tin compounds possessed anthelmintic properties. This was followed by extensive work by Kerr (1952) and Edgar (1956) showing that di-n-butyl tin dilaurate, sometimes referred to as butynorate, is an effective and safe material for the removal of certain species of tapeworms from chickens. The compound was used at the rate of 500 mg per kilogram of feed (0.05 per cent) or as a single 125-mg dose by capsule. The drug is reported as accomplishing removal of the entire tapeworm of the following species: *Railletia cesticillus*, *R. tetragona*, *Choanotænia infundibulum*, *Dacainca proglottina*, *Hymenolepis carioca*, and *Amabotænia sphenoides*.

FLUKES

Flukes are small, flattened, unsegmented worms having a pear-shaped appearance. At least three flukes have been found capable of causing disease in fowls. One of them, *Collyriclum faba*, acts as an external parasite and is discussed in the following chapter (see page 365).

Flukes of the Egg-forming Organs—Flukes of the *Prosthogonimus* species have been found in the cloaca, oviduct and ovary of chickens and ducks, and one of them, *Prosthogonimus macrorchus*, is capable of producing severe injury to the parts invaded by them. This fluke is about $\frac{1}{4}$ inch in length and reddish-brown in color.

Life History—The life history of the parasite is extremely complicated, two intermediate hosts being involved in the cycle. The eggs of the fluke are passed out in the droppings and develop into young worms which penetrate the bodies of snails. Development in the snail takes place with the formation of a tailed form which swims about until it meets and penetrates the body of a dragon fly nymph, in which it becomes encysted. When the dragon fly is eaten by a susceptible fowl, the young fluke is set free, and after migrating to the cloaca of the bird, develops to maturity.

Macy (1934) has published an excellent report in which he discusses in considerable detail the biology of this parasite

Symptoms — Fowls infested with these flukes show dullness, emaciation, sleepiness, and loss of appetite. Kotlán and Chandler (1925) found anemia, emaciation and peritonitis in the affected fowls autopsied by them. There were also many diseased, collapsed ovules which contained grayish-yellow, egg like material mixed with fibrin and pus.

Prevention.—Fowls should be kept away from low, wet areas in which dragon fly nymphs are present.

Treatment — Carbon tetrachloride given in repeated doses of 1.5 cc. is reported by the U. S. Department of Agriculture to have given good results as a treatment for this parasite.

Flukes of the Proventriculus — Newsom and Stout (1933) reported the occurrence in eight weeks-old chickens of a serious disease caused by the presence of a fluke, *Psilostomum ondatrae*, in the proventriculus. The affected chicks showed loss of appetite, stood around with the eyes closed, and gradually wasted away, with death occurring after an illness of several days. At autopsy there was a noticeable enlargement of the proventriculus, with the openings of the secretory glands reddened and inflamed. It was noticed that those chicks which had access to a ditch containing snails were the only birds involved, the other fowls on the place remaining healthy. As soon as the ditch was dried up the snails disappeared and there was no more trouble.

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Chapter 13

EXTERNAL PARASITES

MANY kinds and varieties of external parasites are known to infest poultry. The list includes lice, mites, ticks, fleas, flies and other less important forms. The total loss for which they are responsible is enormous and they present an important practical problem wherever fowls are kept.

LICE

The lice affecting poultry belong to the order *Mallophaga*, chewing lice or bird lice. Over 2000 species of *Mallophaga* have been described, and nearly all of these are found exclusively on birds. Except in the case of domestic poultry, a given species of lice is seldom found on more than one species of host, but a single host may harbor several different species of lice. Some 40 or 50 species of bird lice occur on domestic poultry.

These lice are small, flattened insects, seldom more than $\frac{3}{8}$ inch in length, and yellowish or gray in color. They show considerable variation in details of form and structure, depending upon the particular region of the body which they inhabit. They are not blood-sucking parasites, but have cutting or biting mouth parts with which they feed upon bits of feathers or scales from the skin. In this way, and also by their sharp claws and spiny structure, they cause considerable irritation and annoyance to the fowls on which they live.

They are permanent parasites, the entire life cycle including the egg stage being spent on the body of the host. Heavy infestations frequently occur during cold weather. It is only by accident that lice leave their hosts except to migrate to another host of the same species. Most lice cannot live more than a few days off the body of the living fowl, even when temperature and food conditions are made to approximate those of the host. That the numbers on an individual fowl may be relatively enormous is shown by the reports of

Chandler (1917), who recovered more than 500 specimens from a single hen after fumigation for one and one-half hours to cause the lice to drop off, and of Lamson and Manter (1916) who counted 3600 lice on one chicken, and estimated that they had about one-half of those present. From another chicken one month old, 925 lice were counted. More recently Thompson and Hosking (1937) counted 8270 *Mallophaga* from a single hen. Of this number 92 per cent were the chicken body louse.

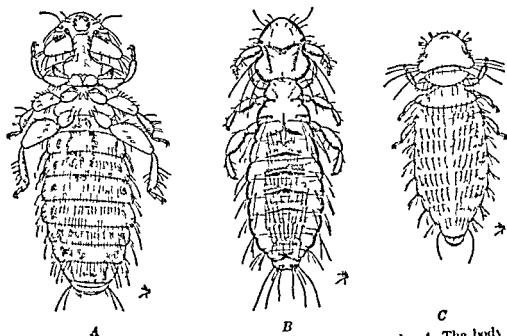


FIG. 79.—Three species of chicken lice, greatly enlarged. A, The body louse *Menacanthus stramineus*; B, the head louse *Cudolopaster heterographus*; C, the shaft louse, *Menopon gallinae*. (After Bischoff and Wood, from the U. S. Department of Agriculture.)

The Common Large Louse.—The chicken body louse, *Menacanthus stramineus**, is one of the most common parasites of poultry. It is most easily located in the region below the vent, although on fowls which are heavily infested it may be found on the breast and back and under the wings.

*The terminology used in designating the scientific names of external parasites is in so far as possible, that approved by the Entomological Society of America.

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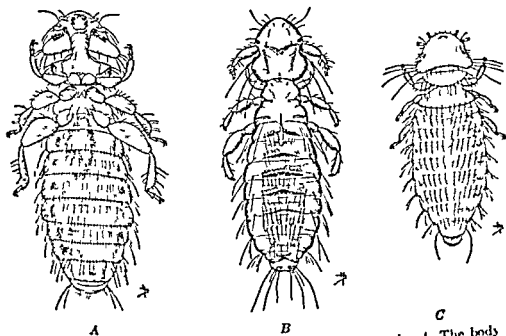


FIG. 79.—Three species of chicken lice greatly enlarged. A, The body louse, *Menacanthus stramineus*; B, the head louse, *Cuculogaster heterographus*; C, the shaft louse, *Menopon gallinae*. (After Bishopp and Wood, from the U. S. Department of Agriculture.)

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It remains on the skin of the host and, because of its great activity, often produces severe irritation of the skin. If the feathers of an infested fowl are separated, these lice may be seen running over the skin in an attempt to get out of sight.

Crutchfield and Hixson (1943) determined the food habits of this and other species by microscopical examination of the crop contents. The body louse not only feeds on barbs and barbules of feathers, but obtains blood by gnawing through the epidermis of the skin and by rupturing quills of pin feathers.



FIG. 80 —Eggs (nits) of the common large louse of the hen

The eggs are deposited in clusters on the base of the feathers, and on badly infested fowls large masses of eggs may be found on the feathers below the vent. The eggs hatch in five to seven days and the young lice mature in about two weeks after hatching. As these lice seem to develop equally well at all seasons of the year, untreated fowls may become very heavily infested at any time. Bishopp and Wood (1917) point out that the heat of the fowl's body is necessary for the hatching of the eggs, and that the lice themselves die in a very short time when off the host. Although primarily a parasite of chickens, this louse is sometimes found on turkeys and occasionally on other fowls running with badly infested flocks of hens.

The Shaft Louse —The shaft louse, *Menopon gallinæ*, is often referred to as the small body louse, but Bishopp and

Wood state that this name does not fit the habits of the species. They point out that it normally is found along the shafts of the feathers, and that it does not remain on the skin of the host for any length of time. Although it is closely related to the large body louse, it is readily distinguished by its habits. It is very common on chickens in most parts of the country, but probably is of much less importance than the large body louse for the reason that it spends most of its time on the feathers rather than on the skin, and seems to feed exclusively on the barbs and barbules of the feathers. In support of this view are the facts that it is not known to occur on young chickens, and that it will live for some time on feathers which have dropped from the fowl. Its life history is similar to that of the body louse except that a longer time is required.

The Head Louse —The chicken head louse *Cuelotogaster heterographus* is found chiefly on the head and neck of chickens and turkeys. It is especially injurious to young birds and is normally the only species likely to cause serious trouble on very young chicks or poults. The adults are dark gray in color about $\frac{1}{8}$ inch long, and are usually found on the top or back of the head, being located so that their mouth parts are close to the skin. The eggs are laid on the down or small feathers of the head, and hatch in five to seven days. The young lice pass through three molts before becoming mature in about thirty days. Wilson (1934) succeeded in rearing this species in an incubator under controlled laboratory conditions with feathers as the only food. The two species just previously described, viz., *Menacanthus stramineus* and *Menopon gallinæ*, failed to thrive under the same conditions.

The Wing Louse —The wing louse, *Ipeurus caponis*, is closely related to the head louse and is the only species commonly found on the large wing feathers of chickens. It does not move about freely and is therefore easily overlooked on colored fowls. It is of much less importance than the three species previously discussed. The slender turkey louse *Oxyporus polytrapezius* is the wing louse of turkeys.

Other Chicken Lice —Other species of lice found on chickens include the large chicken louse *Goniodes gigas* a large, dark gray louse with a head which is broader than it

is long, the fluff louse *Gerresia gallinae* which is a very small light colored species and which is found on the loose fluffy feathers chiefly on the under part of the fowl and the brown chicken louse *Goniodes dissimilis* common in Europe and reported by Bishopp and Wood as probably common in much of the South. No other species yet reported as being found on the fowl has become of much importance.

Lice of Other Poultry—Turkeys, ducks and geese are each infested at times with species of lice that are not found on any other hosts. In the case of the turkey, the large turkey louse *Cleptes meleagridis* is probably the most common. Ducks and geese are rarely infested to the point of serious injury, the slender duck louse *Tricolia crassicornis* and the common louse of the goose *Trinotomanternum* being the ones most often found. Guinea fowl and perfwal are both hosts to specific kinds of lice and the guinea is important as a possible spreader of chicken lice. Neither of these fowls seems to be greatly injured by the lice normally present. Pigeons are often heavily infested with the slender pigeon louse *Columbicola columbae* and less frequently with the small pigeon louse *Campanulotus bidentatus*. Several other species have been reported as occurring on pigeons but the two mentioned cause most of the damage.

Control of Poultry Lice—Development of synthetic organic insecticides has made possible a completely new approach to the control of external parasites of poultry. The long accepted methods involving treatment of individual fowls by dusting, dipping or greasing—though still effective—have been almost entirely replaced by spray or brush treatment of appropriate interior surfaces of the poultry house or by application of insecticides to the floor litter.

Moore and Schwardt (1954) reported on a series of field tests involving more than 200 poultry houses and nearly 60,000 fowls in which some fourteen different organic insecticides were used singly and in combination. They obtained excellent control of the chicken body louse, the shaft louse and the fluff louse with single spray treatments of chlordane, lindane, benzene hexachloride, DDT, lindane, nicotine sulphate-lindane and malathion. Since the flocks used for the tests were selected on the basis of being infested with all

three species, the authors were able to rate the parasites in order of increasing resistance to treatment as the chicken body louse, the shaft louse and the fluff louse.

Insecticide residues and their effects, especially as related to contamination of eggs and poultry meat, place limitations on the use of some of the foregoing materials.

Malathion is both safe and effective when used as a one per cent liquid water spray (1 pint of 57 per cent malathion emulsion in 8 gallons of water) applied to the roosting areas to the point of run-off. It has the further advantage that it will control the red mite or roost mite at the same time with a single spray application.

MITES

There are about twenty species of mites which are known to infest domestic poultry, but only a few of them are sufficiently injurious to be of economic importance. They belong to the family *Acarina* of the class *Arachnida* and therefore are closely related to the spiders. The important species have quite different habits and, in general, a different method of control must be used for each species.

The Chicken Mite.—Most poultry raisers are all too familiar with the common red mite or roost mite, *Dermanyssus gallinae*, and its serious inroads on poultry flocks. Although actually gray in color, these mites usually appear red because they so often contain blood of fowls on which they have been feeding. Since they are blood-sucking mites they can cause serious injury. They are intermittent parasites, hiding in cracks and crevices during the day and coming out to feed on the fowls at night. Because of this habit of getting out of sight during the day, and because they reproduce rapidly, an infestation may easily become severe before the flock owner is aware of their presence unless he is keeping a constant watch for these pests throughout the summer. Their presence is easily determined by examining the ends and under sides of the roosts at the points of support, and by careful inspection of any cracks or crevices in the roosts or roost supports. In severe infestations a characteristic odor is quite apparent.

The English sparrow frequently harbors the chicken mite and according to Fwing (1911) is a most important factor in spreading it from flock to flock. Because of the habit of sparrows in lining their nests with chicken feathers and because their nests are often built in the vicinity of chicken roosts the deserted nests may contain thousands of mites. The mites are also spread by moving fowls from place to place because a few mites may remain on the fowls during the day.



FIG 81 — Roosts should be removable for easy disinfection. Wire beneath the perches as shown here will keep the hens away from the droppings and helps in the production of clean eggs. (Courtesy of Illinois Agricultural Experiment Station.)

Since lice and mites often occur at the same time a single spray treatment with 1 per cent malathion is recommended.

The Scaly Leg Mite — The condition known as scaly leg in fowls is caused by a very small itch mite *Knemidoloptes mutans* less than $\frac{1}{16}$ inch across, which burrows under the scales on the shanks. The severe irritation which is set up by the activities of the mites results in the accumulation of gravish dry debris under the scales the scales being loosened and lifted until the shanks appear to be greatly enlarged. If the condition is left untreated the feet may become deformed and the fowl may get so lame that it has difficulty

in walking or in getting on the roost. The parasite is also found on turkeys, pheasants, partridges and cage birds.

The entire life cycle is spent upon the host. Eggs are laid in the channels formed as the mites burrow in the skin. The young mites pass through a simple metamorphosis and soon start burrows of their own.

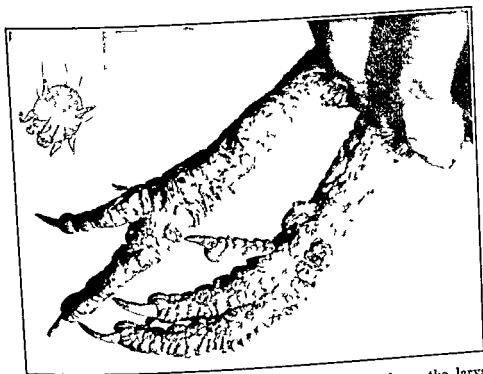


FIG 82 —The condition known as scaly leg. In-set shows the larval stage (greatly enlarged) of the mite, *Knemidokoptes mutans*, that causes the trouble (Courtesy of Illinois Agriculture Experiment Station)

According to Lamson and Manter (1916), there are many remedies for this mite, as any oily preparation with some penetrating power will kill them. One of the best seems to have been originally recommended by Haiduk (1909) and consists of 1 part oil of caraway mixed with 5 parts of vaseline. To gain the most effective results the crusts and scales on the shanks should be softened by soaking in warm, soapy water before applying the ointment. Application of this material by rubbing on the shanks and feet every few days will soon effect a cure.

Where a large flock must be treated it is easier to dip the shanks and feet in a mixture of 1 part of kero-sene and 2 parts of raw linseed oil. This mixture is very irritating because of the kero-sene, and it should be used with considerable care. If the oil gets on the skin it is likely to cause severe burning.

It is very unusual to find this mite in flocks which are kept under conditions of good sanitation, which immediately suggests that a poultryman who follows the principles of hygiene and sanitation and who is careful not to introduce the parasite with purchased stock will not be troubled by it.

The Depluming Mite—Another very small itch mite, *Knemidoloptes gallinae*, may cause considerable injury to fowls. These mites, which are still smaller than the scaly leg mites, live at the base of the feathers, where they produce the condition known as 'depluming scabies'. The intense irritation induced by the mites causes the affected bird to pull out its own feathers. In severe cases the fowls may be nearly bare of body feathers. The large wing and tail feathers are not usually lost.

Fortunately this mite is not of very common occurrence, and when found it yields rather easily to treatment. The oil of caraway ointment just mentioned for scaly leg mites is effective in destroying the depluming mite. Repeated applications of sulphur ointment will also destroy them. The ointment may be made by thoroughly mixing 1 part finely divided sulphur with 4 parts of petroleum jelly or lard.

Bunyea and Wehr (1941) say that complete control may be effected by dipping each bird in the affected flock in a tub of water containing 2 ounces of flowers of sulphur and one-half ounce of laundry soap to each gallon. As the depluming mite is well protected by the skin scales, it is necessary to soak the fowls thoroughly in order to destroy all the mites. Special care should be taken to do the dipping on a warm day. If the flock is also infested with lice, these pests can be eliminated at the same time by adding to each gallon of the dipping mixture three-fourths of an ounce of commercial sodium fluoride.

The Northern Fowl Mite—This parasite, *Ornithonyssus silviarum*, is sometimes referred to as the feather mite, but the true feather mites belong to an entirely different family.

This is a relatively new parasite in the United States, but infestations have occurred in widely separated parts of the country. If left untreated, it causes very serious injury.

In appearance it closely resembles the common roost mite, and may easily be mistaken for it until it is found that the customary methods of treatment are without effect. Because it has many of the habits of both the body louse and the roost mite it is a particularly injurious type of parasite. It lives and reproduces on the fowl and gets its food by sucking blood from the host. The eggs are deposited and hatch among the feathers and the young mites complete their development without leaving the fowl. The mites are most commonly found about the base of the tail and around the vent, and the egg masses commonly occur in the same regions. It often appears that the mites have a decided preference for male hosts, because it is not unusual to find the males in a flock badly infested while the females are comparatively free. Their habit of choosing certain local areas as "feeding grounds" causes the formation of large scabs which spoil the appearance of the dressed carcass. (Payne, 1930)

The parasites may be spread from farm to farm by transporting infested fowls or by wild birds. Several species of wild birds have been found to harbor the parasite, especially purple grackles, rusty blackbirds and barn swallows.

Fortunately there are several methods of treatment which seem to be very effective. One of these is the dip method as used for the depluming mite. A simpler method reported by Payne (1929) is to apply nicotine sulphate to the roosts at intervals of a few days and about twenty minutes before roosting time. Three applications at intervals of three days proved to be 100 per cent effective.

Cameron (1935) found a single application at the rate of 4 ounces for every 100 fowls sufficient in every instance to rid fowls of this parasite. It is important to make certain that all fowls go on the treated roosts. Single birds were effectively hand treated with a few drops of nicotine sulphate smeared around the vent, under the thighs and wings, and on the neck.

Lurman (1932) reported that 10 per cent Neotran dust applied by pinch-dusting or hand-dusting eliminated infestations and protected fowls for a period of twenty-eight days.

Harding (1955) found that 4 per cent malathion dust applied to dry fluff litter at the rate of 1 pound to 20 square feet, with a little mixed into each nest, gave effective control within four days. Heavy infestations on roosters, however, were controlled only by individual treatment with 4 per cent malathion dust.

The Tropical Fowl Mite — This species, *Ornithonyssus bursa*, is very closely related to the northern fowl mite. According to Wood (1920) it is the common mite in Africa, China, India and South America, the roost mite not being found there. In habits it is very similar to the northern fowl mite, except that it is often found in the nests, and breeds there as well as on the fowls. It seems also to be a parasite of the English sparrow, and Wood recommends that a continuous war on the English sparrow be instituted in any locality in which the mite is discovered. Nests should be destroyed by fire, and the nesting places eliminated. Fortunately there have been very few occurrences of this parasite in the United States. The control measures outlined for the northern fowl mite should be effective for this pest.

Chiggers or Red Bugs — Chiggers, *Eutrombicula alfreddugesi*, so annoying to man, may also attack chickens. They are less than $\frac{1}{16}$ inch in diameter and almost invisible, being the first stage of a large red mite, which, when mature, is entirely harmless. Since they are widely distributed in fields, they are readily picked up by chickens on which they attach themselves to the skin in groups. The injury may be very severe in young chickens, and grown fowls are sometimes attacked. Intense irritation is set up, small abscesses are formed at the points where the groups of mites are feeding, and if the exposure is long continued there may be a considerable mortality among young chickens. The chicken chigger, *Neoschongastia americana* is also a pest in the Southern States.

Frequent light dusting of chickens with flowers of sulphur will keep the parasites in check, but the more practical procedure in the Southern and Central States where chiggers are numerous, is to hatch chickens early and to keep them from ranging over areas where the mites are most likely to occur. When chickens do become infested, the application of sul-

MITES

phur ointment as described for the depluming mite, is recommended.

Other Mites of Poultry.—A few of the other mites infesting poultry are of some interest, although they are rarely of economic importance. The tissue mite, or flesh mite, *Laminosioptes cysticola*, bores into the skin and is found in cysts or nodules under the surface of the skin next to the muscles. The mite is exceedingly small, and its presence apparently causes no serious injury.

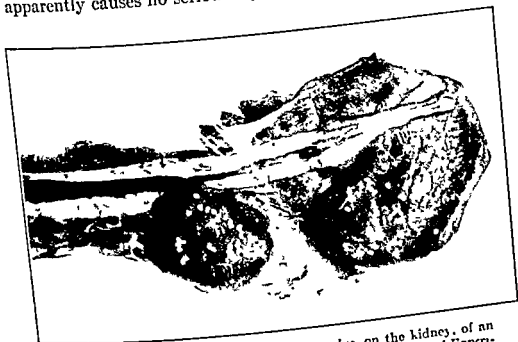


FIG. 83.—Showing air-sac mites, *Cytoleichus nudus*, on the kidney, of an adult fowl. Enlarged about twice (Courtesy of Illinois Agricultural Experiment Station)

The air-sac mite, *Cytoleichus nudus*, occurs in the air sacs and occasionally in other body cavities of chickens and turkeys. Since the air sacs are connected with some of the long bones, the parasite has also been found in the hollow spaces in these bones. It rarely is present in sufficient numbers to cause serious injury, though Hadley (1909) reported 2 cases in which the mites were so numerous as to have perforated the pericardium and the wall of the gall-bladder, thus causing death in a pheasant and in a fowl. It might be argued that this parasite is internal rather than external.

but in any event it is of relatively little importance and it is discussed here because of its entomological relation to other mites

Rebrassier and Martin (1932) state that a quill mite of poultry, *Syringophilus bipectinatus* has been found in five widely separated flocks in Ohio as well as in turkeys and golden pheasants. A specific case is cited in which about 70 per cent of a flock of 1500 fowls were affected with a peculiar molt which in most cases involved over half the body. Examination showed many stumps of quills remaining and in these quills was found a yellowish gray or brownish powder in which the mites were located with the aid of a microscope. No method of treatment or control is known. Another species *S. columbae* occurs on the domestic pigeon.

One of the feather mites *Falculifer rostratus* which eats the barbules and barbicels on wing and tail feathers has been reported on pigeons by Pillers (1927). Fumigation of the pigeons (with heads uncovered) with sulphurous anhydride for two twenty minute periods with an interval of eight days between applications eliminated the parasites.

TICKS AND FLEAS

These parasites are important in the Southern and Southwestern States and may occur infrequently in other parts of the country.

The Fowl Tick.—The chicken tick *Argas persicus* is a serious pest of poultry fowls often being killed by it. The chicken appears to be the favorite host but turkeys geese ducks pigeons guineas and ostriches are all subject to attack. When full grown the tick is from $\frac{1}{4}$ to $\frac{1}{2}$ inch long and it is a powerful blood-sucker. In its feeding habits it is similar to the ordinary roost mite in that it feeds only at night and hides in cracks and crevices or other secluded spots during the day.

The eggs are laid in cracks about the henhouse and hatch in ten or fifteen days if the weather is warm. In cool weather they may not hatch for three months. The young seed ticks which have only six legs crawl up on the fowls where they remain for four to ten days until fully engorged. They then

drop off and secrete themselves as do the adults. In another four to nine days they molt and gain an additional pair of legs. From this time on the habits are very similar to those of adults. The ticks feed only at night and very rapidly. After three alternate feedings and molts the adult males and females appear. The females lay a batch of eggs after each of several feedings. Bishopp (1922) states that each female normally deposits from 500 to 900 eggs. He also points out that the ticks can live for a long time without food, some of the adults in his experimental tests living for two and one half years confined in tight boxes without any food. The seed ticks can live for five or six months, and the nymphal stages for eight to fifteen months without food.

The fowl tick is much more difficult to destroy with chemicals than most other insects and ticks. Because of its flattened shape it is able to crawl into deep cracks so as to be out of reach by ordinary parasitocides. Materials to be effective against the ticks must be penetrating so as to work into cracks and crevices, and they must have lasting power. Carbolineum, a wood preserver containing anthracene oil, is reported by Bishopp as being the most effective material for this purpose. He also states that, "Since the tendency of the tick in all stages is to crawl upward, and not on the ground, it has been found that a roost supported from the floor of the house and kept entirely free from the walls is by far the most satisfactory. It is also essential to have the roost fitted together in such a way that it can be examined or knocked down with greatest possible ease." The construction of nests for easy cleaning and disinfection is also very important.

Bishopp and Wagner (1931) reported that nicotine sulphate diluted with 9 parts of water was effective against the tick when applied to infested poultry houses with a sprayer. It was, however, more expensive and less lasting than carbolineum.

Rodriguez and Riehl (1957) found that 1 per cent malathion spray thoroughly applied under high pressure to the inside and outside structural portions of infested houses and as a fine spray on the floor litter gave complete control of severe infestations by the forty-ninth day after application.

Emmel (1945) observed extreme general weakness amounting to partial paralysis in 14 flocks infested with the fowl tick. Removal of ticks from individual fowls resulted in marvelous recovery, usually within twenty four hours.

The pigeon tick, *Argas reflexus*, is a closely related species which is widely distributed in Europe, but which is not of common occurrence in the United States.

The Sticktight Flea—The chicken flea or sticktight flea, *Echidnophaga gallinacea*, is a serious pest of poultry in the southern part of the United States from Florida to California. The fleas attach themselves to the comb, face, earlobes and wattles, and remain attached by their mouth parts for four to nineteen days. They tend to be found in clusters, the face and earlobes as well as the wattles sometimes being almost black with them. Herrick (1908) records a case in which by actual count there were 164 on the right wattle of a chicken, 65 on the right earlobe and, by estimate, 200 or more on the right side of the face. The pests stood out at right angles to the surface with their heads embedded in the skin. They could not be brushed or scraped off, and even with tweezers they were removed with difficulty.

The flea lays most of its eggs while attached to the host, and according to Parman (1923) they are thrown with considerable force when being passed. Females produce from 1 to 4 eggs a day. The incubation period varies from four to fourteen days, the usual period being six to eight days. The larvæ which Parman observed began to feed on excreta of the adults within a few minutes after emergence and developed rapidly for the first few hours. They were never observed to feed on any other material, and he concludes that the excreta of the adults are necessary for development of the young. The larval period varies from fourteen to thirty-one days.

The mature larvæ spin cocoons of silk and dust and go through a pupal period of nine to nineteen days. During the first days after emergence the adults are inactive, and they do not become attached to a host until after five to eight days. In six to ten days after attachment the females become fully engorged and begin laying eggs. The total minimum period observed from oviposition to the emergence

of the adult flea was thirty days, and the maximum period sixty-five days.

The adults are killed by freezing temperatures and die within a few hours when exposed in tubes in an incubator at a temperature of 100° F. They can live for at least two months without food if the weather is dry and cool, but die very quickly without food if the weather is hot.

Control is rather difficult to accomplish because of the part played by dogs, cats, rats and wild birds in the dissemination of the parasite. In so far as possible these animals should be excluded from the poultry yards. As the dust on the floors of chicken houses and the soil under houses and outbuildings form ideal places for the development of the fleas, special attention must be given to the treatment of these areas. Bunyea and Wehr (1941) recommend that the henhouses and yards be thoroughly cleaned and sprayed with creosote oil. Poultry and animals should be excluded from beneath buildings where the fleas might breed. Infested fowls may be treated by the application of carbolated petrolatum or sulphur ointment, using care not to get any of the ointment into the fowls' eyes.

Emmel (1942) reported that sticktight fleas were completely controlled by feeding 5 per cent of dusting sulphur in the regular mash for a period of three weeks, and scattering sulphur over the litter in the house and soil in the yards, at the rate of 2 pounds to each 100 square feet. Neither feeding nor dusting alone was completely effective.

The European chicken flea, *Ceratophyllus gallinæ*, and the Western chicken flea, *Ceratophyllus niger*, have been reported in Maine and on the Pacific Coast, respectively. Presumably, the same control measures would be effective for these species.

OTHER ECTOPARASITES

Poultry flocks are sometimes attacked by other forms of parasites which do not belong in any of the groups which have been described in the preceding pages. Bedbugs, certain beetle larvæ, cystic flukes and parasitic flies are examples.

Bedbugs.—The common bedbug, *Cimex lectularius*; the closely related European pigeon bug, *Cimex columbarius*; and

the Mexican chicken bug *Hæmatosiphon inodorus* may attack fowls and under favorable conditions may become serious pests. They are rarely found on fowls in the day time but crawl on them at night and suck their blood. Setting hens are often attacked and may be driven from their nests. These parasites hide, breed and lay their eggs in cracks about the walls, roosts and nests. They can usually be eradicated from poultry houses by a thorough spraying with creosote oil or crude petroleum. Since they may easily gain entrance to dwellings and cause much annoyance to human beings, their eradication is doubly important.

Kulash (1947) found that a single application of 5 per cent DDT in kerosene was sufficient to control bedbugs that had heavily infested four poultry houses for over fifteen years. The solution was applied just heavily enough to wet vertical surfaces without any excessive run-off. A careful check of the treated houses one year later revealed no bedbugs nor any evidence of them.

Beetle Larvæ —Bunnea and Wehr (1941) say that there are several different kinds of beetle larvæ which may occasionally attack domestic poultry and that young pigeons are especially liable to attack. They mention the larder beetle *Dermestes lardarius* and the yellow meal worm *Tenebrio molitor* as having been known to attack squabs eating away the skin at the vent and neck, thus producing serious sores. The larvæ of one of the burrowing carrion beetles *Necrophorus* sp. may also attack these young birds.

The adult of the meal worm is said to attack setting hens especially on the feet, causing great annoyance.

Larvæ of the blowflies are sometimes found on domestic fowls, the eggs having been laid by the fly in sores on the fowl or in the natural openings of the body.

Cystic Flukes —Flukes are small flat worms, more or less resembling a leaf in shape. The life history always involves one intermediate host and sometimes two. Two species found in the egg-forming organs and proventriculus have been discussed in connection with internal parasites. Another *Collericium faba* is found in cysts in the skin of chickens and turkeys. It was reported by Riley and Kernkamp (1924) as occurring in young turkeys six to eight weeks old.

which had been reared on a lake shore and in an adjoining marshy meadow. Other turkeys, reared on higher ground without access to the lake were unaffected.

The cysts are smooth and shiny, grayish-white in color, and vary from 2 to 10 mm. in diameter. Most of them show a small black pore through which the eggs of the parasite escape to be scattered wherever the fowl goes. Entire cysts become necrosed and drop off, furnishing additional sources of eggs to be washed into the water. According to Riley (1931), the accumulated evidence points very definitely to snails as the first, and to nymphs of dragon flies as the second intermediate host.

There seems to be no reason to doubt that severe infestations may have a fatal outcome. There is even greater financial loss because of the lowered market value of birds showing conspicuous cysts.

Control of the parasite seems to be readily accomplished by preventing chickens and turkeys from having access to marshy meadows and lake shores where they are likely to feed upon the dragon fly nymphs. Since the English sparrow is sometimes infested with the fluke, as reported by Tyzzer (1918), it may be an important factor in the spread of the parasite.

Black Flies.—Some of the black flies, buffalo gnats, or turkey gnats, as they are variously called (*Simulium* and *Prosimulium* spp.) sometimes become serious pests of poultry. Walker (1927) reports a case in which the death of young goslings was caused by large numbers of black flies which attacked the flock. Gibson (1930) states that they have also caused losses among chickens and turkeys. It appears probable that poultry, as well as larger animals, may sometimes be smothered by drawing great numbers of these small flies into the nasal passages when they appear in swarms, as occasionally happens in the northern parts of the country during the spring.

The eggs are laid on the surface of rocks in swiftly flowing streams or on leaves and stems of plants overhanging such streams. The larval stage, which varies greatly in length according to species, is passed in the water, as is also the pupal stage of one to three weeks. When the adults emerge

they immediately seek warm blooded animals of any kind upon which to feed, though it is of interest to note that it is only the females which suck blood.

Control is extremely difficult, though smudges burned before the doors and windows of poultry houses during the period when the flies are most troublesome will repel them. They will not attack in the smoke.

Mosquitoes—Surveys in Alabama by Edgar and Williams (1948) revealed that the common pest mosquito, *Culex quinquefasciatus*, has a high preference for the blood of the domestic fowl. Over 99 per cent of the engorged females whose blood could be identified by serological or microscopical methods were found to have fed on avian blood. *Culex quinquefasciatus* was the only species captured in a chicken baited trap and specimens of this species were found most often in chicken houses rather than in adjacent stock barns.

Control is probably most easily accomplished by means of DDT sprays.

The Pigeon Fly—This parasite, *Pseudolynchia canariensis* has been known in the United States since 1896, according to Bishopp (1929) but became especially troublesome during the summers of 1928 and 1929. It is not only a serious pest of pigeons but it is a necessary agent in the transmission of pigeon malaria. In squab picking and packing rooms the flies are often a source of annoyance to the workmen, but under normal conditions they do not bite man.

Drake and Jones (1930), and Coatney (1931) have described the fly and its habits. It is a blood-sucking fly and is not able to live away from the host for any great period of time. Its feeding habits greatly annoy the squabs, fledglings and older birds. The adult fly remains almost constantly upon the pigeon and is not known to feed upon other domestic animals or wild birds.

The fly is viviparous, the mature larvæ (pre-pupæ) being deposited singly by the female. Bishopp states that deposition of the larvæ occurs for the most part while the flies are on the pigeons and that the pupæ are thus concentrated in the nests. When first deposited the full-grown larva is enclosed in a whitish capsule or pupal case and is incapable

of locomotion. Transformation into the resting stage begins immediately, and within three hours the capsule or puparium becomes somewhat shiny and jet black in color. The pupal stage lasts about thirty days. Soon after the fly emerges it is ready for a meal of pigeon's blood, which seems to be essential to its further existence. Coatney found that the flies could live on chicks and mourning doves, but these birds do not harbor the parasite under natural conditions. His

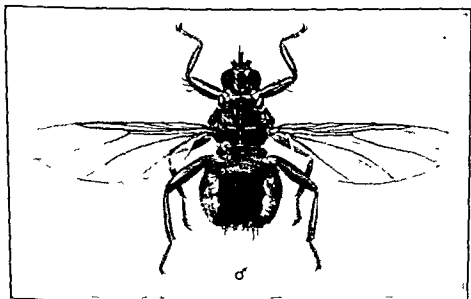


FIG. 84.—The pigeon fly, *Pseudolynchia canariensis*. (After Drake and Jones.)

experiments on man indicated that the flies feed about twice every twenty-four hours, but the females were not able to live and produce larvæ when fed exclusively on human blood.

Control of the parasite consists first in cleaning out and disposing of all manure, dirt and nesting materials so as to insure death of the pupæ. Since the pupal stage lasts about thirty days, this cleaning should be repeated at intervals of not more than twenty-five days. A minimum of twenty days would be safer.

Bishopp found that 1 to 3 pinches of pyrethrum powder dusted among the feathers killed all flies on squabs. It was less effective on older birds.

Drake and Jones report the complete eradication of flies from a large pigeon farm in Iowa by the use of thorough clean-up measures combined with the dipping of all pigeons in pyrethrin-soap solution. The material for the dip consisted of 86.5 ounces of sodium oleate, 9.5 ounces of the alcoholic extract of pyrethrum flowers, and 6 ounces of sodium silicate. The mixture is non-poisonous and is used at the rate of 1 ounce of the concentrate in each 6 gallons of soft water. Over 2000 pigeons were dipped on one farm during warm, sunny days in the first part of October without the loss, or even apparent injury, of a single bird.

Yager and Gleiser (1946) found that DDT, 10 per cent in talc, on one trial proved very effective in the control of the pigeon fly as well as the slender pigeon louse, *Columbicola columbx*. The material was dusted on at the rate of 3 grams per pigeon.

Since many pigeon lofts are still free from these pests, all breeders who are shipping stock should see to it that such birds are free from flies before shipment. A purchaser who wishes to be certain of not introducing the parasite, would do well to give any incoming birds a thorough dusting with pyrethrum powder before placing them with his own stock.

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APPENDIX

DRUGS

INDIVIDUAL treatment of sick fowls is rarely beneficial or profitable. The old adage that an ounce of prevention is worth a pound of cure is even more true in combating diseases of fowls than in dealing with the ailments of other farm animals. There are however several drugs which have been very effective in flock treatment of fowls affected with certain diseases and parasites. Reference has been made to them in preceding chapters but a descriptive list of some of the more common drugs is included here for convenient reference.

Ildrin — The common name for a complex naphthalene compound used as an insecticide. Available as a dust and as an emulsifiable concentrate. Must be used with care because it is readily absorbed through the skin and is highly toxic. Not often recommended in poultry practice.

Antibiotics — Antibiotics are widely used in human medicine as well as in the treatment of certain animal diseases usually by intramuscular injection. In low concentration usually not more than 5 milligrams per pound of feed they act as growth-stimulating factors for chickens and turkeys. Hundreds of antibiotics have been isolated from various sources and many of them are highly effective against various bacteria and some viruses. Among those in common use are aureomycin (chlortetracycline) bacitracin, penicillin, streptomycin and terramycin (oxytetracycline).

Argyrol — A vitellin silver preparation is a compound of silver and protein containing about 30 per cent of silver. It occurs as dark brown flakes, is soluble in water and is used in a 5 to 20 per cent solution. Argyrol solution is non-irritating and may be used in the eyes or nose as an antiseptic.

Boric Acid — A white crystalline powder which may be used alone or in combination with tannic acid or iodoform as a dusting powder for wounds. The watery solution of boric acid (it is slowly soluble at the rate of about 5 parts of boric acid to 100 parts of water) is useful as a wash for

inflamed eyes and mucous membranes Boric acid is a weak antiseptic and has a soothing protective action when applied to raw surfaces

Carbon Tetrachloride — A colorless liquid having a sharp, penetrating odor It has been recommended as a vermifuge against some of the roundworms of fowls The dose for a mature fowl is 1 cc, and it is best given in a gelatin capsule The limitations of this and other medicinal agents for expelling intestinal parasites have been presented in Chapter 12

Chlordane — The common name for a chlorinated hydrocarbon insecticide which is effective against lice, ticks, and fleas Acts as a stomach poison as well as by contact and as a fumigant Must be used with care

Derris Root — The powdered root of any one of several species of plants commonly known as derris It is effective against chicken lice when used as a dust in 20 per cent concentration with precipitated calcium carbonate (chalk) or other carriers It may also be employed for this purpose in the form of a dip, using $\frac{1}{4}$ ounce of the powder to 1 gallon of water

DDT (*Dichloro-diphenyl-trichlorethane*) — This highly effective insecticide is a crystalline white solid, practically odorless and tasteless, insoluble in water but soluble in most organic solvents It is especially effective against flies, but is characterized by slow "knockdown" and kill The residual effect is such that walls and ceilings sprayed with oil emulsions (such as 2 per cent DDT in deodorized kerosene) will give 100 per cent kill a month later to flies confined in the room over night

For use on the poultry farm it is available not only as a dusting powder, but with added wetting agents which permit ready suspension in water For ordinary use against flies or mosquitoes, a concentration of 2.5 per cent DDT in water is recommended

Dieldrin — The common name for a complex naphthalene compound used as an insecticide It is chemically related to aldrin It must be used with care to avoid inhalation, skin contact and food contamination

Di-n-butyl tin dilaurate (*Butynorate*) — A tetravalent tin

compound which has been found to be an effective anthelmintic against the fowl tapeworms *Raillietina cesticillus*, *Choanotenia infundibulum*, *Dactinea proglottina* and *Hymenolepis cariosa*.

Enkeptin — A brand name for 2-amino-5-nitrothiazole. It is used in the control of blackhead (enterohepatitis) of turkeys.

Epsom Salts (Magnesium Sulphate) — Occurs as small whitish or colorless needles and is widely used as a physic. Administration is best accomplished by dissolving 1 pound of the crystals in about 2 gallons of water, this being the dose for 100 mature fowls. If all water is withheld until about 8:30 A.M. the fowls will quickly drink their fill of the treated water, which may be replaced by fresh water at the end of an hour's time.

Ferric Chloride — Commonly used in the form of a tincture, or alcoholic solution, which is dark brown in color and stains tissues with which it comes in contact. It is useful in reducing the amount of bleeding from minor wounds, and it is sometimes used for that purpose following cropping of the wattles or dubbing of the comb.

Furazolidone — One of the nitrofurans made synthetically from furfural. It is effective against fowl typhoid, hexamitiasis, infectious sinusitis, synovitis and enterohepatitis.

Iodine — Tincture of iodine is an alcoholic solution of iodine and potassium iodide. It is dark brown in color and has a pungent, alcoholic odor. It is useful as an antiseptic when applied to wounds. A wound so treated should not be dressed or bandaged because of danger of severe burning.

Iodoform — A yellow, crystalline substance containing about 96 per cent of iodine, and having a strong, penetrating odor. It may be used alone or in combination with boric acid or tannic acid as a dusting powder for wounds. When placed in contact with tissues iodoform slowly releases iodine, and it is to this property that its antiseptic action is attributed. Because of its penetrating odor, care should be used to keep it away from all food products.

Kamala — A brownish, odorless powder obtained from the capsules of a small tree indigenous to the East Indies. Though of doubtful efficacy, it is often used as a vermifuge.

against tapeworms It may be purchased in capsules or in tablet form, and is administered in 1-gram doses to mature chickens Caution should be used in giving kamala to turkeys, as it is apparently more toxic for them than for chickens

Malathion — One of the phosphorus-containing insecticides which has proved highly effective against lice and mites of poultry, as well as against the northern fowl mite and the fowl tick

Mercurial Ointment — A dark blue ointment which, as ordinarily sold, contains 30 per cent of mercury It is very effective for destroying lice on fowls and is applied by spreading a small amount over the skin just below the vent For economy and ease of application it is customary to dilute the rather heavy, stiff ointment with 1 or 2 parts of vaseline The amount of vaseline to add will vary with temperature conditions, the larger quantity being suitable only for cold weather use

Naphthalene — This drug occurs as white, shining flakes and has a strong coal tar color It is a fairly good insecticide and, if sprinkled around the henhouse, aids in reducing the number of insect pests When finely ground and mixed with twice its weight of vaseline, it is very effective as an ointment to be applied in small amounts around the tail and vent to destroy the northern fowl mite If used as the active ingredient in a louse powder, care should be taken not to use it in more than 20 per cent concentration When used in 60 per cent concentration, and well rubbed in, it has proved fatal to hens

Nicarbazine — A complex anticoccidial agent consisting of 4,4'-dinitrocarbamilide and 2-hydroxy-4,6-dimethylpyrimidine Low levels in the feed afford protection against coccidia while permitting normal growth and feed conversion Hens which normally lay brown eggs will after a few days of nicarbazine feeding lay eggs which are almost white Shell color returns to normal within a few days after medication is stopped

Nicotine Sulphate — Commercially available as a solution containing 40 per cent of nicotine It is extremely poisonous if taken internally and should be handled with caution for that reason It is effective as a fumigant against lice, and is

used by applying it to the top surface of the perches by means of a brush or small oil can, using 8 ounces to each 100 feet of perch, just before the fowls are allowed to roost. The effect of the material is not lasting, and a second application in eight to ten days is essential to complete eradication of the lice.

Nitrofurazone — One of the nitrofurans made synthetically from furfural. It is effective against both cecal and intestinal coccidiosis.

Paradichlorobenzene — Occurs as white crystals, soluble in alcohol but insoluble in water. With a melting-point at 127° F., it is volatile at ordinary temperatures. It may be substituted for naphthalene in an ointment used to destroy the northern fowl mite, particularly when fowls must be treated in cold weather.

Phenothiazine — A light yellow, crystalline powder, insoluble in water but slightly soluble in mineral oils. Used in dosages up to 0.5 cc. for the control of cecal worms. Reported to be ineffective against the large intestinal roundworm.

Potassium Permanganate — Consists of dark purple crystals which have a metallic sheen. A freshly-made solution of this drug in water has a reddish color, which is gradually lost upon exposure to air. Because oxygen is released when the solution comes in contact with organic matter, potassium permanganate has an antiseptic and deodorizing action. It has been widely used in the drinking water for fowls, but because it soon becomes inert as a result of rapid deoxidation, it probably has little real value in this connection.

Sodium Chloride (Common Salt) — A physiological solution of common salt is made by dissolving 8.5 grams in 1000 cc. of distilled water. For practical purposes the solution may be made by adding 2 level teaspoonfuls of salt to 1 quart of boiled water. It is used for washing and cleansing wounds. Because its osmotic pressure is the same as that of blood, no change is induced in the tissues with which it comes in contact.

Sodium Fluoride — Occurs as a white powder or as clear, lustrous crystals. The drug is poisonous and is irritating to the eyes and mucous membranes. Sodium fluoride is effective against the lice of poultry, and may be used as a dust or in

solution as a dip As a dip to rid fowls of lice the solution is made by adding 1 ounce or slightly less of sodium fluoride to each gallon of warm water

Sodium Fluosilicate —A white, odorless, granular powder It is effective against poultry lice, and may be used in the same manner as sodium fluoride

Sulfa Drugs —A group of drugs of the sulfonamide type which have a marked bacteriostatic action Some are also definitely coccidiostatic They are used medicinally to enable the immune bodies of the host to combat the invading organisms

Included in the list are sulfanilamide, sulfamerazine, sulfamethazine, sulfapyridine, sulfathiazole, sulfaguanidine and sulfaquinoxaline In general they are soluble in dilute acid or acetone, insoluble in ether, and only slightly soluble in water Sodium-sulfamerazine and sodium-sulfamethazine are water soluble

Sulfa drugs must be used with caution since many of them are sufficiently toxic, even when fed at very low levels, to interfere with normal growth and egg production, and with calcium deposition in egg shell formation

Sulphur (Orchard-spray Type) —A light yellow powder having very little odor Sometimes used as a dust against poultry lice, but is not so effective as sodium fluoride Sulphur has been found effective for destroying the depluming mite of fowls, and for this purpose it is used in a dip consisting of 2 ounces of finely divided (wetable) sulphur and 1 ounce of laundry soap in each gallon of water

Tetrachlorethylene —A colorless, heavy fluid having a penetrating, ether like odor It is very similar to carbon tetrachloride, but in many respects appears to be superior to that drug It has been found to be of some value when used as an anthelmintic against some of the roundworms of poultry The dose for an adult fowl is 1 cc, and it is best administered in gelatin capsules

Tobacco —Tobacco dust consists of the finely powdered leaves of the tobacco plant It has been found to be fairly effective for expelling the large roundworms and cecal worms of fowls The dust to be used for this purpose should contain not less than 1.5 per cent of nicotine It is administered by

including it in the dry mash at the rate of 2 per cent over a period of three to four weeks

DISINFECTANTS

On every well managed poultry plant there is frequent regular and almost constant need for a reliable and effective disinfectant

By a disinfectant is meant a substance which is capable of killing microorganisms. A complete disinfectant is one which not only destroys microorganisms in the forms in which they usually occur, but which kills the more resistant spore forms as well. A distinction should be made between disinfectants and antiseptics. The latter are substances which will inhibit or prevent the growth of microorganisms without necessarily destroying them.

Two factors enter into the choice of a disinfectant, viz, efficacy and cost. An inferior disinfectant is expensive at any price, because the germ killing power of the material is the primary reason for its use. Fortunately there are available at reasonable prices several substances which have known germicidal properties.

Bichloride of Mercury — This substance occurs as heavy, colorless crystals or as a white powder, and is odorless. It is extremely poisonous, is a powerful disinfectant even in dilute solutions, and is corrosive to metals. It is not suitable for use in wounds because it coagulates proteins and is irritating to tissues. When applied in a dilution of 1 to 1000 to clean surfaces, bichloride of mercury is useful as a disinfectant.

Carbolic Acid (Phenol) — Pure carbolic acid at ordinary temperatures occurs as long, whitish crystals, having a characteristic odor. For convenience it is usually sold in liquid form by the addition of a small amount of water. Phenol is sometimes used as a disinfectant in 5 per cent solution and in that strength it is a reliable germicide, but it is more expensive than some of the other agents which are equally efficient.

Phenol is commonly used as the standard with which to compare other disinfectants in determining their efficacy. Concentrations are chosen which will kill a standard culture

of *Escherichia coli* (*B. coli*) in two and one-half minutes. The phenol coefficient of a disinfectant is then expressed as the parts by weight of pure carbolic acid equivalent to 1 part of the particular disinfectant.

Chlorinated Lime (*Calcium Hypochlorite*) — This material often incorrectly called chloride of lime, is a white powder having a strong odor of chlorine. It is an efficient disinfectant in 5 per cent solution (6 ounces to a gallon of water), and in addition it possesses marked deodorizing properties.

Creolin — A dark brown liquid, having a sharp, characteristic tar like odor. It forms a milky solution with water, and is sometimes used in a 4 or 5 per cent solution as a disinfectant.

Cresol — A straw-colored fluid having a tarry odor. It gradually turns dark with age. Cresol does not go into solution readily unless warm soft water is used, and since there is danger in using a mixture in which the cresol is not completely dissolved, care should be taken to mix thoroughly. The disinfectant property of this material is dependent upon its cresylic acid content, and no grade below that guaranteed to contain at least 90 per cent of cresylic acid should be used. In a 2 to 3 per cent solution (approximately 1 quart to 12 gallons of water), cresol is a reliable disinfectant.

Formalin (commonly called *Formaldehyde*) — This is a 40 per cent solution of formaldehyde gas in water. It is a clear, colorless liquid having a pungent, irritating odor. Formalin is very penetrating and is a powerful disinfectant when used in 8 to 10 per cent solution (equivalent to 3 to 4 per cent of formaldehyde gas). Formalin should be kept tightly stoppered to prevent escape of the gas, with consequent loss of strength. Because of the irritating effect of formalin, care should be taken not to let it come in contact with the skin. The eyes, especially, should be protected.

Liquor Cresolis Compositus (*Compound Solution of Cresol*) — A dark brown syrupy liquid having a strong coal tar odor. It is composed of equal parts of cresol (U.S.P.) and linseed-oil-potash soap. In a 3.5 to 4 per cent solution, (approximately 1 quart to 7 gallons of water) it is an efficient disinfectant, and it has the advantage of mixing readily with water.

Saponified cresol solutions as prepared by various manufacturers are often used as substitutes for compound solution of cresol. Under the regulations of the United States Department of Agriculture, only such saponified cresols as meet certain requirements are permitted for official disinfection of cars, boats, premises, etc. A list of such permitted disinfectants is revised at intervals, and may be obtained on request from the Bureau of Animal Industry, Washington, D. C.

Lye.—When used in 2 to 3 per cent solution, lye is an efficient germ destroyer. It has the advantage of being relatively cheap and readily available. Because of its caustic action, care should be exercised when applying the solution not to get it in contact with the eyes or face. The use of lye in the treatment of fowls infested with tapeworms has been discussed in Chapter 12.

Quaternary Ammonium Compounds.—These compounds are widely used as disinfectants because they act fast, and are odorless, nonirritating and noncorrosive. They are also good deodorants. Like other disinfectants, they should be applied only to properly cleaned surfaces. They are much less effective in hard water than in soft. The usual concentration is 200 p.p.m. and at this strength they are effective against most of the disease-producing bacteria and viruses.

Sodium Hypochlorite.—A grayish-white powder which has a marked chlorine odor, and which is somewhat unstable when exposed to air. It is used as a disinfectant in the same manner (5 per cent solution) as calcium hypochlorite, to which it is very similar. (There are on the market certain hypochlorite powders which are effective in more dilute solutions.) The chlorine disinfectants may be used about the premises and in the poultry buildings without danger of imparting undesirable odors or flavors to eggs or other food products.

Sodium Orthophenylphenate.—This is a new disinfectant, the value of which has recently been demonstrated by workers in the U. S. Department of Agriculture. It is readily soluble in water and is therefore easy to use, but to be effective it must be applied at a temperature of 60° F. or above. When properly applied, it has been found to be highly effective against the tubercle bacillus and against the virus of infectious

laryngotracheitis. It has only a slight odor, is not productive of undesirable flavors in food products, and is relatively non-toxic for livestock and humans. It is, therefore, especially adapted as a disinfectant for use in poultry feeding stations. It should be used in a 1 per cent solution.

The Use of Disinfectants.—When using any disinfectant, the following suggestions should be observed:

1. To be efficacious, a disinfectant must be of the proper strength.

2. The area to be disinfected must be clean, so that the disinfectant may penetrate well.

3. Either an old broom or a spray pump may be used. The advantage of the latter is that it insures greater penetration into cracks and crevices, and offers greater protection to the operator.

4. Since most disinfectants are very irritating, the operator should protect the exposed parts of his body, particularly the eyes, from contact with the solution.

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